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EDITORS
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HEART MURMURS

PART II

BY

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From the Cardiac Department of the London Hospital

Received August 28, 1947

A phonocardiographic study of the innocent heart murmurs and those found in mitral stenosis was described in an earlier publication (*Brit. Heart J.*, 1947, **9**, 1). In this paper the murmurs appearing in aortic valvular disease, hypertension, congenital heart disease, heart block, and in anæmia are examined.

III. THE MURMURS OF AORTIC VALVULAR DISEASE

The finding of a systolic murmur in the mitral area in patients with disease of the aortic valve is commonplace. Often it is difficult on clinical grounds to decide the source of the mitral murmur, whether it comes from the aortic valve, whether it is caused by the hypertrophied left ventricle, or whether it arises from associated disease of the mitral valve. Indeed, so loud is the mitral murmur in many patients with aortic valvular disease, especially stenosis, that it has seemed natural to assume that the mitral valve is also affected; cardioscopy has then been applied to decide whether mitral stenosis accompanied it or not, but often this examination has been inconclusive.

A phonocardiogram was recorded in 40 cases of aortic valvular disease from the mitral area for it was to the elucidation of the mitral systolic murmur that the investigation was primarily directed. Often a record was taken from the aortic area as well, but as it added no exceptional information, routine recording in this area was not pursued. Among the 40 cases there were 10 with aortic stenosis, 20 with aortic incompetence, and 10 with probably aortic valve sclerosis. The phonocardiographic findings in each group will be described in turn.

Aortic Stenosis

Not all the cases of aortic stenosis showed a thrill, but each had a loud and rough systolic murmur both in the aortic and mitral areas. The presence or absence of diastolic murmurs in both these areas was noted. The electrocardiogram was often abnormal; on cardioscopy a varying degree of left ventricular enlargement was found, and as a rule the aortic shadow was abnormal, commonly from unfolding of the aorta.

The mitral systolic murmur in the phonocardiogram started at the S line in five cases, immediately after the S line in three, and a little later in another (Fig. 21 and 22). In one case where the murmur started before the S line there was a mid-diastolic murmur as proof of the association of mitral stenosis. The systolic murmur always continued up to or within a short distance of the second heart sound.

In 2 of the 10 cases an early diastolic murmur of aortic incompetence was heard with the stethoscope although the systolic murmur was more in evidence. The remaining 8 were regarded as uncomplicated aortic stenosis because the murmur of incompetence had not been found although it had been sought diligently by direct auscultation during halted respiration

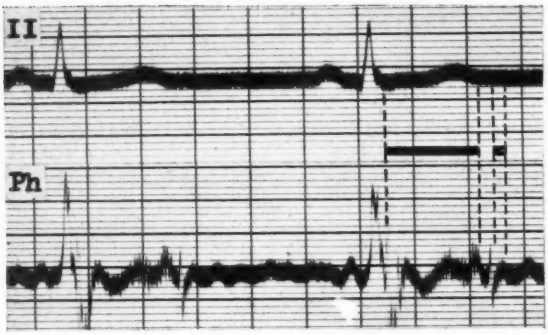

FIG. 21.—Aortic stenosis. The systolic murmur starts at the S line, and a diastolic murmur following the second heart sound is evidence of accompanying aortic incompetence.

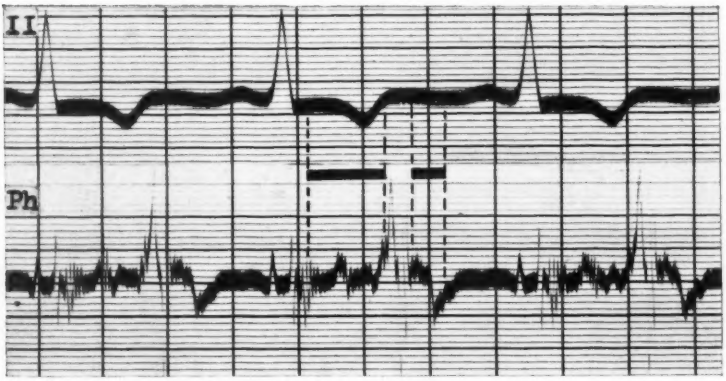

FIG. 22.—Aortic stenosis. The systolic murmur starts a little later than the S line. A diastolic murmur follows the second heart sound, giving proof of aortic incompetence, although this murmur was not heard on auscultation.

and with the patient in the upright posture; yet in all eight of them a murmur immediately following the second heart sound was recorded in the phonocardiogram proving the presence of aortic incompetence.

AORTIC INCOMPETENCE

There were 20 cases of aortic incompetence in which a rheumatic origin could not be substantiated on clinical grounds although the comparative youthfulness of five supported that ætiology. Radiological examination too could not establish definitely the presence of mitral disease; in the right oblique position the left auricular impression was usually prominent but it was difficult to tell with certainty whether this was caused by a displacement backwards of a normal left auricle due to the enlarged left ventricle, or caused by a left auricle distended from mitral disease.

In 8 cases the aortic incompetence was considered to be of syphilitic origin, but in 4 of these the phonocardiogram showed auricular and mid-diastolic murmurs proving a rheumatic ætiology. In all these 8 cases an Austin Flint murmur had been suspected by others.

but in the 4 where the sound record proved the presence of mitral stenosis the murmur was evidently a true presystolic murmur. Of the other 4 cases with alleged Austin Flint murmur the phonocardiogram showed nothing distinctive in two apart from the early diastolic murmur, and neither auricular nor ventricular moieties of the first heart sound showed anything odd (Fig. 23). In two cases where the aortic regurgitation was free the murmur lasted throughout diastole and was continued into systole (Fig. 24). In these four non-rheumatic cases the systolic murmur commenced at the S line (Fig. 25).

In the 12 cases where the aortic incompetence was considered to be non-luetic, there was no clinical evidence of mitral disease that would have suggested a rheumatic origin for the aortic lesion, but in every instance the phonocardiogram supplied such a proof, demonstrating both auricular and mid-diastolic murmurs. It is true that in two patients, aged 17, and in three, aged 26, 28, and 28 years respectively, a rheumatic aortic incompetence had been presumed because of their age, but the phonocardiographic test gave the proof.

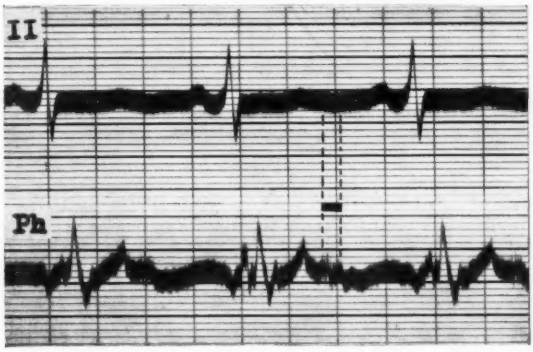

FIG. 23.—Luetic aortic incompetence. A diastolic murmur accompanies the second heart sound. Although three observers told of the presence of an Austin Flint murmur on auscultation the auricular and ventricular moieties of the first heart sound show nothing distinctive.

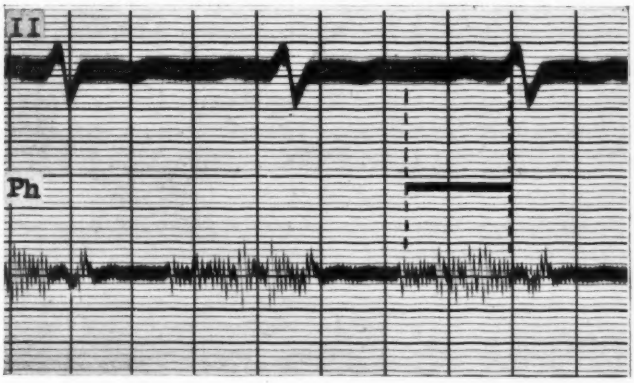

FIG. 24.—Aortic incompetence, luetic in origin and of considerable degree. The diastolic murmur starts at the second heart sound and is continued through the whole of diastole into systole.

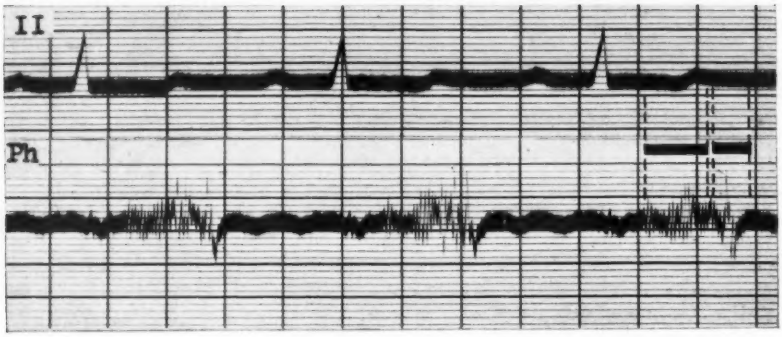

FIG. 25.—Aortic incompetence. The systolic murmur in this case starts in mid-systole some distance after the S line.

The early diastolic murmur was always recorded immediately after the second heart sound, and although variable in its duration, it was usually short and had spent itself mostly when the third heart sound was reached, except when the regurgitation was prominent it lasted throughout diastole to reach auricular systole. In this event the phonocardiogram cannot help to decide the ætiology of aortic incompetence; it is only when the early diastolic murmur has stopped short of auricular systole that an auricular systolic murmur will indicate mitral stenosis, and should it stop short of the third heart sound a mid-diastolic murmur is added as a sign of mitral stenosis.

It is likely that the phonocardiograph, when it comes into routine use, will often reveal an undiscovered aortic incompetence by recording the early diastolic murmur (Fig. 26), although when the test has shown it to be present, careful re-auscultation should discover the murmur.

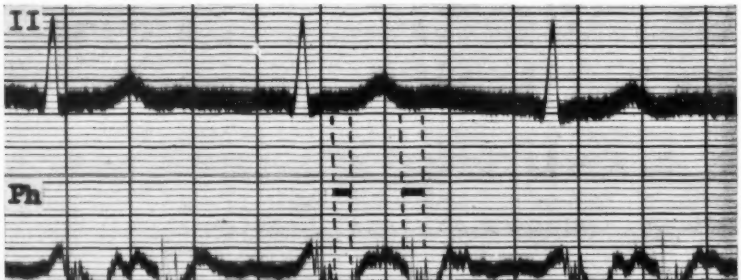

FIG. 26.—Early aortic incompetence. Clinical examination had concluded that a systolic murmur was of the innocent kind, but the phonocardiogram showed an early diastolic murmur. Subsequent examination by direct auscultation easily confirmed the presence of this murmur.

Having noticed the frequency in elderly subjects of a mitral systolic murmur where an aortic systolic murmur is heard best or only heard in the upright posture on direct auscultation and unaccompanied by a thrill, I collected 36 such cases. The cause of the murmur seemed to

be a benign one, and so it proved in two of the patients when examined at necropsy; in one, death had resulted from carcinoma of the bronchus, and in the other, from an unusual form of anæmia. The aortic cusps in both cases showed bars of thickening at their bases by calcareous atheroma which had spread from the aorta on to the aortic aspect of the cusps, producing a sub-clinical stage of aortic stenosis (*aortic valve sclerosis*). A phonocardiogram was recorded in the mitral area in 10 such cases. In 6 the systolic murmur started at the S line as it had done in most patients with aortic stenosis and aortic incompetence, but in the remaining 4 it was postponed until mid-systole (Fig. 27). In 6 cases the murmur stopped short of the second

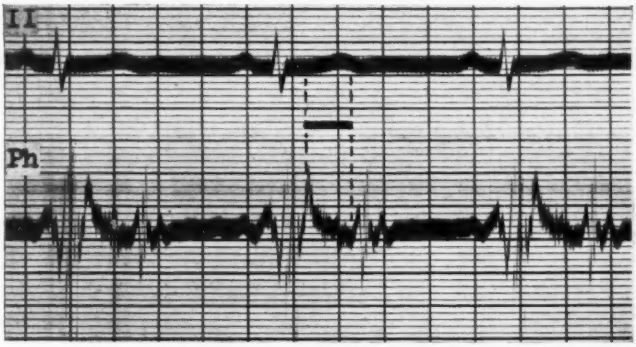

FIG. 27.—Aortic valvular sclerosis. The systolic murmur in this case starts in mid-systole a short distance beyond the S line.

heart sound, and in this respect it differed from the systolic murmur of aortic stenosis and incompetence which reached the second heart sound.

SUMMARY: MURMURS OF AORTIC DISEASE

The phonocardiographic investigation of murmurs heard in the mitral area in *aortic stenosis* has shown that the clinical diagnosis may have to be corrected in two ways. First, the commoner revision applied to the additional diagnosis of aortic incompetence when this has gone unsuspected by auscultation; this added finding was present in the phonocardiogram in every case in this series as it happened, but I have found exceptions since. Secondly, when aortic valvular disease had been regarded as a lone lesion the phonocardiogram twice showed auricular and mid-diastolic murmurs at the apex proving that mitral stenosis was present. Whenever the aortic lesion existed alone the systolic murmur usually started at the S line which marks the commencement of ventricular systole.

In the case of *aortic incompetence* the phonocardiogram frequently demonstrated the presence of mitral stenosis where this had gone unrecognized on clinical and radiological examination. In half the cases where the aortic incompetence was believed to be luetic in origin and showed a murmur that might be described as Austin Flint, the sound tracing showed it was a presystolic murmur of mitral stenosis; in the other half the sound record either showed no change in the auricular or ventricular moieties of the first heart sound, or the early diastolic murmur had lasted throughout diastole into auricular systole.

IV. THE MURMURS OF HYPERTENSION

Patients in whom hypertension was associated with some other form of heart disease were excluded, and only those with hypertension alone were admitted for special study. A phonocardiogram was recorded in 43 cases: in 16 it was used to tell the position of the added sound

that was initiating triple rhythm; in 14 to examine the auricular and ventricular moieties of the first heart sound which showed splitting on auscultation; in the remaining 13 to examine a systolic murmur in the mitral area that was a noticeable auscultatory sign.

The Systolic Murmur

Routine clinical and radiological examination failed to explain with certainty what determined the presence of a systolic murmur in hypertension. The murmur did not disallow a triple heart rhythm because they were sometimes found together in the same patient. The murmur was not a product of associated aortic valvular disease, although in a group of patients, not included in this paper, where aortic incompetence accompanied hypertension, the mitral area was never without a systolic murmur. In hypertensive heart failure a mitral systolic murmur was common although certainly not invariable, but it was noticed that all patients with this murmur showed considerable enlargement of the heart. Thus, among the factors contributing to an apical systolic murmur in hypertension, cardiac enlargement was the most obvious. Further, when a number of patients with a systolic murmur and hypertension, but with only a moderate degree of cardiac enlargement, were examined by the phonocardiograph, mitral stenosis was found to be present although the signs of this had been inconclusive on clinical and radiological examination.

On auscultation the murmur was always distinct, and often it was loud. It never disappeared on deep inspiration, and posture had little effect on it except that often it was heard best in the reclining posture. Being loud the murmur was heard some distance away from the seat of its maximum intensity in the mitral area. There was no selective direction for its propagation, and it was never directed specially towards the aortic area. A regard of the physical qualities of the murmur could not tell it from the systolic murmur of mitral disease, although the absence of a thrill justified the suspicion that it belonged to hypertension, especially if enlargement of the heart was considerable. Radiological examination most times could exclude the presence of added mitral disease and yet a prominent left auricular impression in the barium swallow in the right oblique position often called for care in interpretation. A low and distinctive curve in the left oblique position supported a diagnosis simply of hypertension where a normal left auricle had been displaced backwards by the enlarged left ventricle.

In each of the 13 cases of hypertension with an apical systolic murmur, the phonocardiogram showed the murmur starting in mid-systole, a little way beyond the S line (Fig. 28).

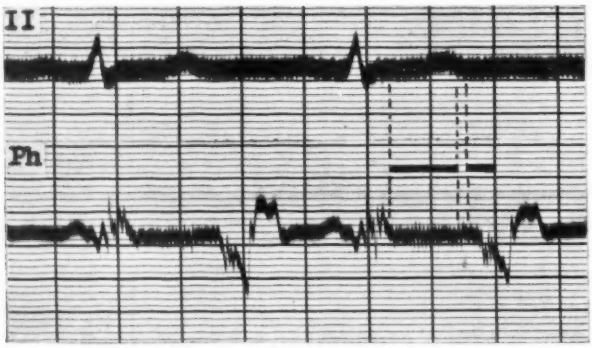

Fig. 28.—Hypertension. The systolic murmur starts in mid-systole a little beyond the S line. A diastolic murmur accompanying the second sound continues in this case up to a prominent third heart sound.

It sometimes lasted up to the second heart sound although occasionally the murmur had spent itself before the end of systole. This was in sharp contrast with the murmur of mitral disease which never once started later than the S line and generally started in front of it and during the P–R period of the electrocardiogram, that is, during auricular systole. Apart from the mid-systolic position of the murmur, the phonocardiogram of hypertension differed from that of the tracing of mitral valvular disease in that there was no mid-diastolic murmur. In the presence of *auricular fibrillation* the same features distinguished the phonocardiogram of mitral disease from that of hypertension, so that in hypertension the systolic murmur started in mid-systole and some way after the S line and there was no mid-diastolic murmur (Fig. 20) whereas the systolic murmur in fibrillation from mitral stenosis started at the S line and a mid-diastolic murmur followed the third heart sound (Fig. 29). The start of the systolic murmur of hypertension in mid-systole also distinguished it from cases of aortic valvular disease where the murmur in most instances started at the S line.

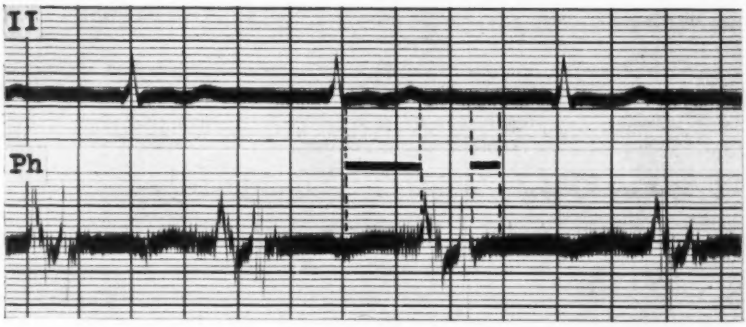

FIG. 29.—Mitral stenosis and auricular fibrillation. The systolic murmur starts at the S line and a diastolic murmur follows the third heart sound.

THE DIASTOLIC MURMUR

Among 43 cases of hypertension there were 12 whose phonocardiogram showed an early diastolic murmur (Fig. 30), although in none could this murmur be elicited by auscultation. The significance of these vibrations of high frequency (as with a murmur) following and contiguous with the second heart sound in the phonocardiogram has been examined, and the

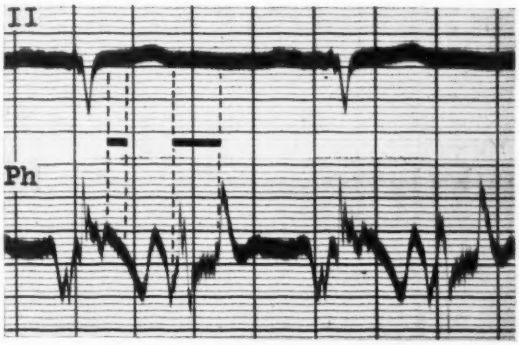

FIG. 30.—Hypertension. A diastolic murmur, which was not heard clinically, accompanies the second heart sound.

possibility considered whether the cause lies with the sudden distension of the aorta in hypertension during early diastole and not with the presence of relative aortic incompetence. In support of a degree of aortic reflux being present were the facts that such a sign was often absent from the sound tracing in cases where the diastolic blood pressure was high and in others where the second aortic sound was very loud, and the presence of the same murmur (or its phonocardiographic pattern) in aortic stenosis where the second sound was not loud and where the diastolic blood pressure was not raised.

A diagnosis of *mitral incompetence* has been applied when a mitral systolic murmur is prominent among the physical signs of hypertension. In 13 patients with hypertension who showed a mitral systolic murmur, it never once coincided with the start of ventricular systole so that it was not produced by incompetence of the mitral valve.

Summary: Murmurs of Hypertension

The commonest murmur in hypertension is an apical systolic murmur. Cardiac enlargement above all else contributes to its appearance. Should a patient with a raised blood pressure, therefore, show a systolic murmur in the absence of more than moderate left ventricular enlargement, it is likely that the murmur arises from a source other than hypertension. The murmur starts in mid-systole and later than the S line in the phonocardiogram so that its mechanism is not mitral incompetence. An early diastolic murmur from relative aortic incompetence was often recorded in the sound tracing although it could not be heard, but this graphic finding seems to be of secondary importance.

V. THE MURMURS OF CONGENITAL HEART DISEASE

In this variety of murmurs too, phonocardiography has proved useful in confirming a diagnosis when there was doubt and in excluding the innocent kind. The conditions in which the phonocardiogram was most used were patent ductus arteriosus, pulmonary stenosis, auricular septal defect, ventricular septal defect, and coarctation of the aorta.

Patent Ductus Arteriosus

A sound record was taken in 11 cases of undoubted patent ductus arteriosus and a distinctive murmur was found in each. Clinically the murmur was loud and of the machinery kind and was accompanied by a thrill. The third heart sound was never identified on

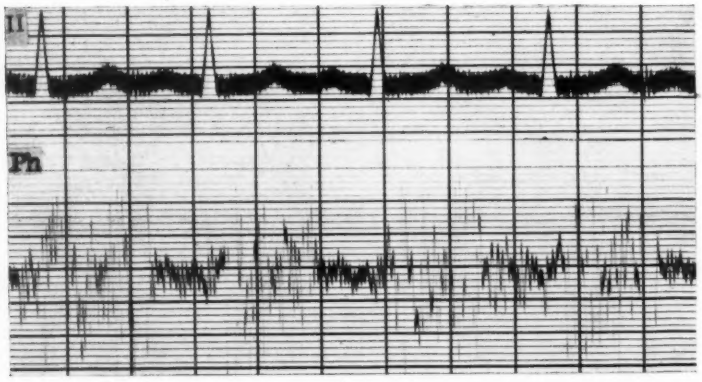

Fig. 31.—Patent ductus arteriosus. The murmur starts at the S line and soon increases, but it declines in mid-systole to increase again in late systole and reaches its height at the second heart sound which it embraces; it also obscures the third heart sound, and occupies the rest of diastole as a moderate murmur.

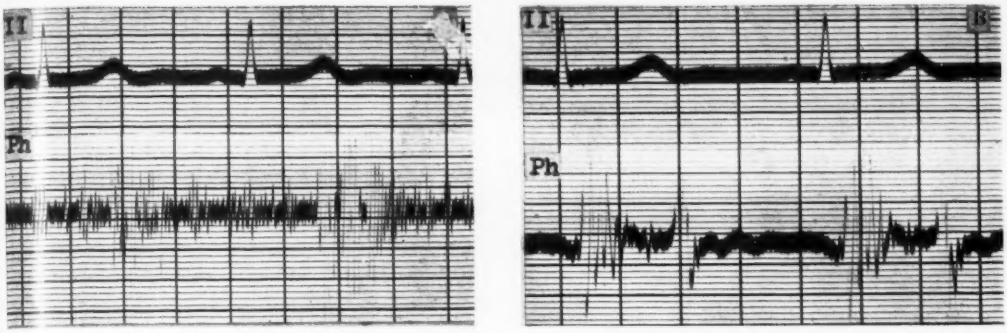

FIG. 32.—Patent ductus arteriosus. The characteristic continuous murmur present in (A) is absent in (B) which was recorded after the duct was ligated.

auscultation amongst the murmurs. Phonocardiographically the murmur was found to be as distinctive as it was clinically, its main characteristic being its continuity throughout systole and diastole. Indeed, when tracing this murmur the galvanometer string is never still (Fig. 31 and 32). The murmur in early ventricular systole starts at the S line, and a little later it becomes more obvious; in mid-systole the murmur wanes; in late systole the murmur increases in intensity at a point corresponding with the summit of the T wave in the electrocardiogram, and at the end of the T wave, where the second heart sound occurs, the murmur is at its height. After thus embracing the second sound it declines until it reaches the position of the third heart sound which it also obscures. Thereafter it continues as a subdued murmur throughout the remainder of diastole and auricular systole. This graphic finding explains the difficulty met with in some cases of patent ductus arteriosus of discovering the second heart sound within the murmur during auscultation.

Pulmonary Stenosis

A phonocardiogram was recorded in 14 cases; in 11 of these the pulmonary stenosis was considered to be a lone lesion, while in the remaining 3 there was also present a defect of the ventricular septum (Fallot's syndrome). In the first group the murmur in the pulmonary area was loud, and sometimes harsh, and was accompanied by a thrill. In the second group it was less loud and a thrill was absent twice. In both groups the murmur, although louder in the reclining posture, was easily heard in the upright posture. The second heart sound was present as a rule, and no diastolic murmur was heard in these patients.

The murmur in early ventricular systole commenced at the S line; in mid-systole the murmur waned; in late systole it usually increased in intensity at a point coinciding with the beginning of the T wave of the electrocardiogram, until it reached the second heart sound which it embraced, but it ceased before the third heart sound was reached. The relative intensity of the murmur in early and late systole varied so that sometimes it was louder in early systole (Fig. 33), and at other times it was louder in late systole (Fig. 34).

The Innocent Pulmonary Systolic Murmur

A systolic murmur in the pulmonary area is a common finding in young subjects, and since so little has been spoken about the differentiation of the innocent murmur from that of pulmonary stenosis, it is a common cause of unwarranted invalidism. Phonocardiography has contributed materially in differential diagnosis because it has confirmed the validity of the clinical signs that identify this innocent murmur.

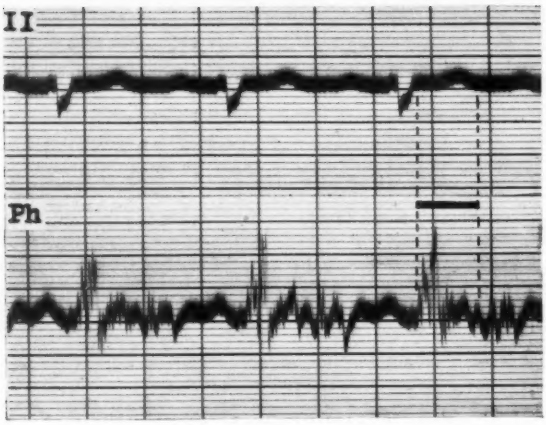

FIG. 33.—Pulmonary stenosis. The systolic murmur starting at the S line is more intense in early ventricular systole; it embraces the second sound, but it is spent before reaching the third heart sound.

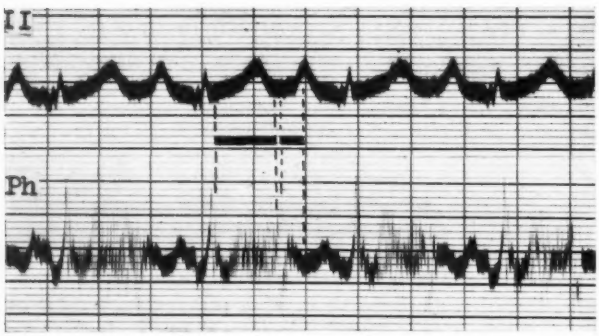

FIG. 34.—Pulmonary stenosis. The systolic murmur which starts at the S line is more intense in late ventricular systole; it overlaps the second sound but disappears before the third heart sound.

The murmur was rough in quality and sometimes it was moderately loud. The effect of posture was a valuable test, for the murmur was much less noticeable in the upright posture, especially when combined with deep inspiration. This innocent murmur was never accompanied by a thrill although the absence of this sign could not be accepted as evidence of the innocence of the murmur since it was often absent when pulmonary stenosis was associated with ventricular septal defect. These clinical signs could not be controlled by reference to the electrocardiogram nor to cardioscopy in both of which negative findings at such tests did not mean that pulmonary stenosis could be excluded, although positive findings were naturally evidence of the organic lesion.

The phonocardiogram will prove a valuable aid to the diagnosis, and in the meantime it has supported the physical signs that have been regarded as the basis of a clinical judgment on the innocence or otherwise of the common systolic murmur in the pulmonary area. The distinctive feature of the sound record was that the murmur started in mid-systole

a little later than the S line and had largely spent itself before reaching the second heart sound (Fig. 35); naturally there were no diastolic murmurs present.

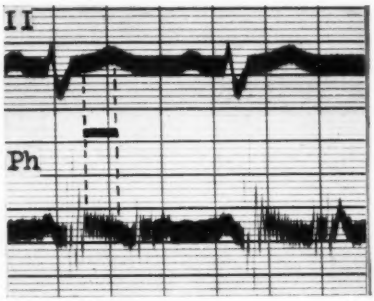

FIG. 35.—The innocent murmur in the pulmonary area. The murmur starts in mid-systole and it has spent itself before reaching the second heart sound.

AURICULAR SEPTAL DEFECT

When a cardiological examination includes an electrocardiogram and cardioscopy, this congenital defect will seldom be missed because of the characteristic signs that these will show. Clinically, however, its recognition continues to remain uncertain. The pulse is usually small, the apex beat is out, and a systolic murmur is seldom absent along a line from the base to the apex, but greatest reliance is placed on finding an early diastolic murmur in the pulmonary and mitral areas which is probably not heard to the right of the mid-line as is the murmur of aortic incompetence. When such a murmur is present—and it has a high incidence—moderate or greater dilatation of the pulmonary artery can be predicted on radiological examination for it is the outcome of relative pulmonary incompetence from dilatation of the arterial ring. If a clear third heart sound is heard along with this murmur the diagnosis of auricular septal defect is more certain.

The auscultatory findings are shown characteristically in the phonocardiogram (Fig. 36) with the systolic murmur starting at the S line, the early diastolic murmur following immediately after the second heart sound, and a clear third heart sound beyond. This phonocardiographic finding is in contrast with the early diastolic murmur of rheumatic aortic incompetence when the third heart sound, if present, is often followed by the mid-diastolic murmur of mitral

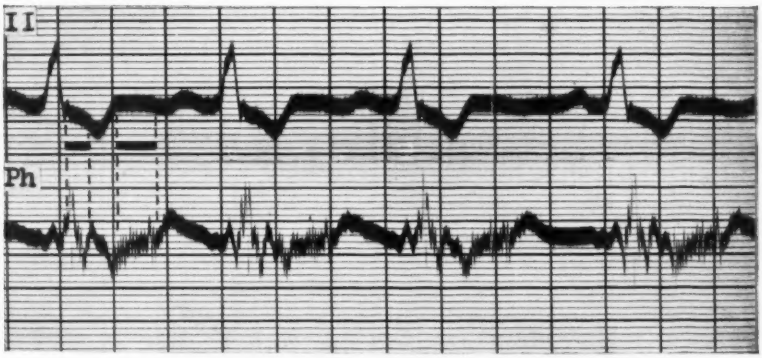

FIG. 36.—Auricular septal defect. The systolic murmur starts at the S line and the diastolic murmur of pulmonary incompetence accompanies the second heart sound and lasts as far as the prominent third heart sound.

stenosis. Occasionally, however, auricular septal defect is present alongside mitral stenosis (Lutembacher's syndrome) where in addition to the early diastolic murmur of pulmonary incompetence there are the auricular and mid-diastolic murmurs of mitral stenosis (Fig. 37).

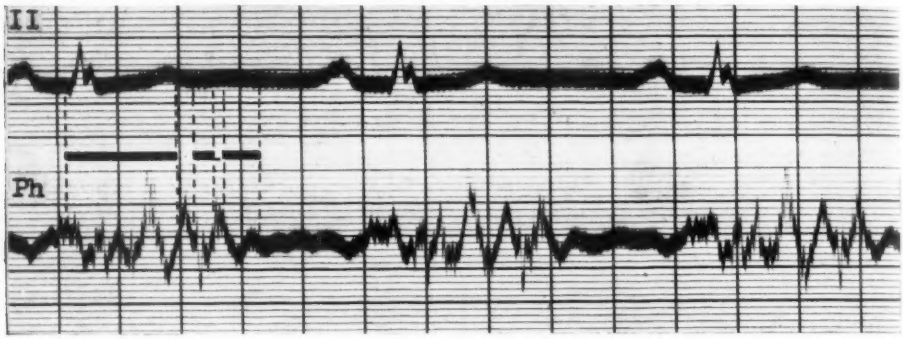

FIG. 37.—Auricular septal defect and mitral stenosis (Lutembacher's syndrome). The systolic murmur of mitral stenosis starts during auricular systole and a mid-diastolic murmur follows the third heart sound. The diastolic murmur following the second heart sound is caused by pulmonary incompetence.

VENTRICULAR SEPTAL DEFECT

The murmur of ventricular septal defect has already been described in the section dealing with the innocent parasternal murmur. It is loud and almost invariably accompanied by a thrill. Apart from the possible association of an abnormal electrocardiogram and radiogram, the phonocardiogram is characteristic for it shows the murmur starting at the S line and, therefore, earlier than the innocent murmur; it also lasts longer (Fig. 8 and 9 in previous paper).

COARCTATION OF THE AORTA

Hardly any of the orthodox areas for auscultation of the heart are without a systolic murmur in a patient with coarctation of the aorta, although it is seldom loud anywhere. Like the murmur over the back, the murmur over the front of the chest usually arises from the hypertrophied arteries of the collateral circulation. Rarely, the murmur has its source in the heart from the greatly enlarged left ventricle, and especially in the presence of aortic incompetence.

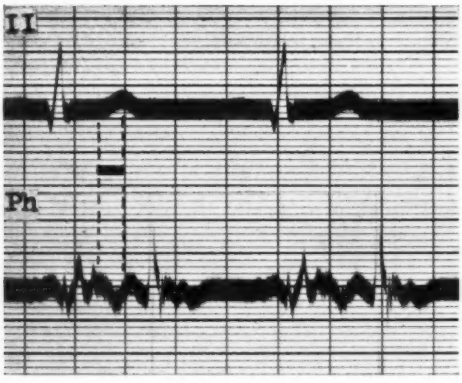

FIG. 38.—Coarctation of the aorta. The systolic murmur starts in late systole and some distance after the S line.

In the second circumstance, the phonocardiogram shows the murmur commencing in mid-systole, but if it arises in the hypertrophied arteries it is even later in systole (Fig. 38). The incidence of relative aortic incompetence, producing an early diastolic murmur in the sound record, is commoner in coarctation hypertension than in other forms of hypertension.

Summary: Murmurs of Congenital Heart Disease

The murmur of *patent ductus arteriosus* is a continuous one lasting throughout systole and diastole, with intensification in early diastole where it covers the second heart sound and proceeds to obscure the third heart sound as well.

The murmur of *pulmonary stenosis* starts at the S line and lasts till it reaches the second heart sound which it embraces, but it ends before reaching the third heart sound. The murmur appears earlier in systole than the innocent pulmonary murmur, commencing as it does at the S line of the phonocardiogram.

An early diastolic murmur from pulmonary incompetence followed by the third heart sound are the characteristic phonocardiographic findings in *auricular septal defect*. When mitral stenosis is added to the congenital anomaly (Lutembacher's syndrome) there are added auricular and mid-diastolic murmurs.

The systolic murmur of *ventricular septal defect* commences at the S line and thus differs from the innocent parasternal murmur which starts in mid-systole; it also lasts longer.

The common systolic murmur in *coarctation* is the one heard over the hypertrophied arteries of the collateral circulation so that it occurs late in systole. Less frequently the murmur comes from the greatly enlarged heart and aortic incompetence is then usually present, so that the murmur is earlier in systole.

VI. THE MURMUR OF HEART BLOCK

A systolic murmur in the mitral area in heart block is only found when the heart is greatly enlarged. It is a rough murmur and it may be loud. Posture is without much effect on its intensity although it is sometimes better heard when the subject inclines to the left. A thrill is never present unless there is mitral or aortic valvular disease as well.

In seven cases of complete heart block with a systolic murmur in the mitral area, the phonocardiogram showed the murmur starting in mid-systole (Fig. 39). Graphically it was

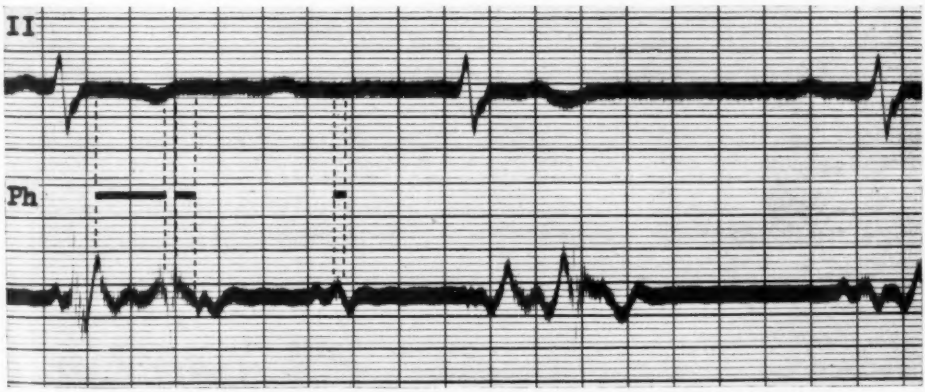

Fig. 39.—Complete heart block. The systolic murmur starts in mid-systole, and a diastolic murmur follows the second heart sound. The murmurs are similar to those found in hypertension associated with much cardiac enlargement. The natural auricular diastolic murmur is also recorded.

similar to the murmur of hypertension except that the tracing did not as often show an early diastolic murmur which was sometimes present in the record from a case of hypertension. Although hypertension appeared along with complete heart block in three cases, the systolic murmur was in the same situation in the other four without hypertension.

Summary: Murmurs of Heart Block

The systolic murmur of complete heart block has been shown by the phonocardiogram to start in mid-systole. Thus, the mechanism of the murmur in heart block as well as of the one in hypertension appears to be connected with the muscular contraction of a much enlarged heart; the start in mid-systole excludes a valvular origin for the murmur.

VII. THE MURMUR OF ANÆMIA

Although an obvious murmur is heard in the majority of patients with a significant anæmia, it is sometimes absent even when the anæmia is severe. Its presence does not depend on the size of the heart. The murmur as a rule is widely distributed and may be heard in the aortic area as well as in the pulmonary and mitral areas; when present in more than one area it is usually loudest in the pulmonary. The murmur varies in intensity so that it may sound faint or rough, and it is better heard in the reclining than the upright posture. Although diastolic murmurs have been described in anæmia, I have never heard them.

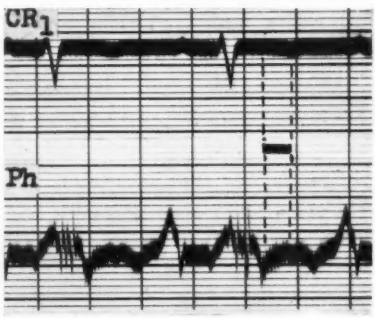

Fig. 40.—Anæmia. The systolic murmur starts in mid-systole some way after the S line. A prominent third heart sound follows the second.

Whenever the phonocardiogram was recorded it showed the murmur starting in mid-systole (Fig. 40). An obvious third heart sound was seen whenever the heart was enlarged as the result of the anæmia.

Summary: Murmurs of Anæmia

The only murmur to show in the phonocardiogram from patients with anæmia was the one in mid-systole. A diastolic murmur was not once recorded.

General Summary and Conclusions

The heart murmurs in 500 healthy subjects and patients with heart disease were specially examined, both by clinical auscultation and by phonocardiography. The object of the investigation was to determine the physical characteristics of murmurs that would make their clinical recognition both easier and more certain. Ancillary methods of examination including electrocardiography were used whenever necessary, and cardioscopy was never omitted. Necropsy was sometimes available.

Foremost amongst the findings of the investigation was the clinical identification of the *innocent mitral systolic murmurs* which continue to cause widespread unwarranted invalidism because of their common appearance; on this account their recognition is a matter of concern unequalled by any other in clinical cardiology. The situation and distribution of these murmurs and the effects upon them of a change in posture and deep breathing make it possible to tell them from the murmurs of organic heart disease. The phonocardiograph confirmed this clinical classification and located the murmurs either in mid-systole or late systole; it also showed the absence in such cases of auricular systolic and diastolic murmurs. In patients with *mitral stenosis* the phonocardiograph showed that the murmur which presented clinically as a systolic murmur usually started during the P–R period of the electrocardiogram, that is during auricular systole, as did the presystolic murmur. Even in the few exceptions where the murmur was later and coincided with the start of ventricular systole, the term " mitral incompetence " would have been incomplete as in each there was a mid-diastolic murmur giving proof of mitral stenosis. The common incidence of a mid-diastolic murmur immediately succeeding the third heart sound in mitral disease, for it was found in each of 74 cases, stimulates the clinical search for this murmur in patients suspected of the condition. Should future investigation show that this is an invariable graphic finding in mitral disease it will prove to be a physical sign of inestimable value.

In *aortic valvular disease* the systolic murmur, which was an unfailing sign in the mitral area, was seen phonocardiographically to start synchronously with the start or early part of ventricular systole. The test showed the common incidence in aortic stenosis of the early diastolic murmur of aortic incompetence although this sign had not yielded to clinical auscultation. The phonocardiograph has often demonstrated an early diastolic murmur when aortic incompetence is unsuspected by casual or even careful clinical examination. On this account too the test has proved of great value.

The mitral systolic murmur in *hypertension*, making its appearance whenever the heart is greatly enlarged, was recorded in mid-systole and a little time after the ventricle had commenced to contract; such a finding opposes the assumption that this murmur is the outcome of mitral incompetence. Doubtless it is the result of muscular contraction and the same murmur was found when the heart was much enlarged in *complete heart block*. In both conditions an early diastolic murmur of relative aortic incompetence, resulting from dilatation of the aortic ring, was a fairly common event, and when it was present it served to emphasize the presence of considerable left ventricular enlargement.

The phonocardiogram in *congenital heart disease* helped to tell the innocent parasternal murmur from that of ventricular septal defect, and the innocent pulmonary systolic murmur from that of pulmonary stenosis. The test demonstrated the continuity of the murmur of patent ductus arteriosus with its accentuation in early diastole. An early diastolic murmur from pulmonary incompetence, leading up to an obvious third heart sound, was never absent in the sound tracing from cases of auricular septal defect; when mitral stenosis was added to the congenital lesion (Lutembacher's syndrome) so were auricular systolic and mid-diastolic murmurs to the phonocardiogram. The murmur of coarctation of the aorta was written in late systole. In *anæmia* the murmur started in mid-systole and a diastolic murmur was not once recorded.

This phonocardiographic study of heart murmurs has shown that the quality and intensity of murmurs matter far less than their place in the cardiac cycle and in relation to the heart sounds. Such a finding emphasizes the need for self-catechism during clinical auscultation when precise answers should be sought to six set questions. The questions concern the character of the first heart sound and similarly of the second heart sound, the presence of more than two sounds, and the presence of murmurs connected with the first heart sound

(presystolic and systolic), with the second heart sound (late systolic and early diastolic), and with the third heart sound (mid-diastolic). When such simple auscultatory procedure has become custom, cardiological diagnosis will have gained immeasurably in accuracy.

Dr. John Parkinson, Physician to the Cardiac Department, has given me his advice in the preparation of this paper. Mr. William Dicks, Chief Technician to the Department, has helped me with the phonocardiograms.

REPETITIVE PAROXYSMAL TACHYCARDIA

BY

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Paroxysmal tachycardia might be defined as sudden attacks of extreme acceleration of the heart that last from a few seconds to a few days and end abruptly as they begin. In the average patient with paroxysmal tachycardia the paroxysm is the exception: normal rhythm is the rule; the difficulty is to obtain an electrocardiogram during the attack. We have become interested in a special variety where recurrent paroxysms are the rule and normal rhythm is the exception; the difficulty now is to obtain the normal electrocardiogram. This variety is here described as *repetitive*. It is characterized by recurring short runs of auricular, nodal, or ventricular extrasystoles, runs or paroxysms of tachycardia in fact, almost constantly present for months or for years and only occasionally interrupted by the normal sinus rhythm. We do not include under this term the recurrent brief paroxysms of tachycardia which may happen to be portrayed on an isolated electrocardiogram. It is true that in a sense they are repetitive, but this is an incident, not a clinical state like that of repetitive paroxysmal tachycardia where the play of paroxysms almost displaces normal rhythm. Its separate consideration is justified on clinical grounds; for it more often affects children and young people in whom it presents a novel problem in prognosis. There are few conditions in children of which it can be said that they are likely to " grow out of it," but this appears to be one.

It is 20 years since one of us (J. P.) came across our Case 1, and since then we have records, more or less complete, of 40 cases of repetitive paroxysmal tachycardia. Cases to be quoted below have been reported singly or perhaps in twos or threes. Our plan was to place on record for the first time a large group of such cases. We have since found, however, in the monograph of Léon Gallavardin (1946), a study of this nature under the title " Extrasystolie auriculaire en salves," differing from ours in that it relates to auricular paroxysms only.

Lewis (1909) made the first observation of this kind in a man of 41 with an otherwise healthy heart. By continuous polygraphic record during 109 minutes he was able to count 54 short paroxysms of auricular paroxysmal tachycardia at a rate of 133–184. Paroxysms of over 1 minute duration were rare; the shortest attack consisted of a succession of a few ectopic beats, the longest lasted 7 minutes. Though occurring at rest, they were more frequent on exertion. Even at a slow rate the pulse was hardly ever regular because of single or multiple auricular extrasystoles.

White (1920) drew attention to the " markedly rapid but not immediate " increase in rate and similar offset during the short and continuously recurring attacks of auricular paroxysmal tachycardia in his patient observed for five years. Scott (1922) abolished with quinidine the short and continuously recurring attacks of ventricular tachycardia which were precipitated by exertion and were the cause of serious invalidism in this patient.

Louis Gallavardin (1922, *a*, *b*, and *c*) insisted on their distinction from ordinary paroxysmal tachycardia and described the ventricular variety in one case (Gallavardin, 1922*b*) and the auricular variety in three others (Gallavardin, 1922*c*), under the name of " Extrasystolie

á paroxysmes tachycardiques." Later with Dumas (Gallavardin and Dumas, 1924) he used the name of " tachycardie en salves " in adding three more supraventricular cases. He also noticed in some that effort produced the paroxysms (Gallavardin, 1922*a*) and that the ventricular form frequently caused attacks of giddiness comparable with those in Stokes-Adams disease. Later with Veil (Gallavardin and Veil, 1929*a* and *b*) he added four similar cases which also include the one already published in 1922; after 15 years of observation the patient was still having the short and continuously recurring paroxysms of ventricular tachycardia.

Cassidy (1924) recorded an " incessant " paroxysmal tachycardia in an otherwise healthy child of 8, who had short bursts of auricular paroxysmal tachycardia every few minutes, constantly recurring during two years of observation.

In Gilchrist's (1925–6) Case 5 with ventricular tachycardia mostly on exertion " an intimate mixing of short ventricular paroxysms, multiple and isolated ventricular extrasystoles and occasional sinus beats " were continuously recorded for six months until they were abolished with quinidine.

Wenckebach and Winterberg (1927) reported two instances of almost continuous auricular paroxysmal tachycardia with varying A-V block, watched for two and three months respectively. In three other cases short bursts of ventricular paroxysmal tachycardia were constantly found, and in two of these effort produced longer attacks. They conclude that it would be better to distinguish a persistent form of paroxysmal tachycardia from the usual one occurring in attacks, as one does in flutter.

Single cases of the ventricular variety were described by Jones and White (1926–7), Schliephake and Graubner (1928), Lutembacher (1929), Andersen (1931), and Bérard (1931). In Gallavardin and Froment's (1930) case salvos of ventricular tachycardia co-existed with auricular paroxysmal tachycardia of the classical type. In McMillan and Bellet's (1931–2) patient, also with the ventricular form, a Cæsarean section was successfully performed while she was in the state of paroxysms. Wilson and others (1932) in adding four similar cases insist on the absence of structural heart disease, the relation of the paroxysms to exertion, their brevity, and the effectiveness of quinidine. This was also successfully employed in other ventricular cases quoted above.

Froment (1932) dedicates part of his admirable monograph on ventricular paroxysmal tachycardia to the " Extrasystolie ventriculaire bénignes á paroxysmes tachycardiques." He draws a sharp distinction between these and the " grave," " terminal," or " prefibrillatory " ventricular tachycardias and insists that the denomination of ventricular paroxysmal tachycardia should be reserved for these only while the former benign condition should be called extrasystolia. Transitional forms, however, are described where prolonged observation was necessary before placing the case into the one or the other group. Diagnostic criteria are given in favour of " extrasystolia," namely, the uniform shape of the extrasystolic complexes forming the tachycardiac runs, the normality of the intervening sinus beats, the youth of the patient, the integrity of the cardiovascular system, and the long evolution of the arrhythmia. Twenty cases are reported from the literature and no original cases are added.

The prognosis in general has been reported good. An exception was the 56-year-old patient of Clerc *et al.* (1933) with habitual ventricular tachycardia who died suddenly. Then, Elliott and Fenn (1934) described a 29-year-old woman in whom ventricular paroxysmal tachycardia with shorter or longer intermissions lasted for more than three months, after which she died of congestive heart failure. Each of these patients had an enlarged heart and an abnormal cardiogram between the attacks. Maddox (1947) has added a case in which there was a sudden mortal termination. Routier and Puddu (1936) and Routier (1937), describing three cases of benign ventricular extrasystoles in the form of paroxysmal tachycardia, also stress the good prognosis, provided there is no cardiac enlargement and the cardiogram is normal between the attacks. Campbell and Elliott (1939) also believe that the type " of very short

paroxysms recurring frequently, may be found with a normal heart and seems to be of no serious significance, even though it is ventricular."

Fine and Miller (1940) describe an " orthostatic " auricular paroxysmal tachycardia observed for two years, in whom the attack could be produced at will by change of posture, though it was also found sometimes at rest and in the supine position. Four years later the patient, who had congenital lues, died suddenly during malaria therapy (Miller and Perelman, 1945). On the other hand a similar patient, a healthy girl of 14, with " chronic auricular tachycardia " at rest and especially on change of posture, was able to live an active life during three years of observation (Miller and Perelman, 1945). Peters and Penner (1946) describe an orthostatic paroxysmal ventricular tachycardia successfully treated with quinidine. Graybiel and White (1946), in commenting on a cardiogram of recurrent auricular paroxysmal tachycardia, find it decidedly uncommon and believe that it is " in itself no evidence of heart disease."

Léon Gallavardin (1946) in an exhaustive study of the auricular extrasystole has collected 31 cases of " extrasystolie auriculaire en salves," partly original, partly previously published by his father (Louis Gallavardin) and others, with observation periods ranging from a few months to 29 years. In two-thirds of the cases the arrhythmia was the only cardiac abnormality while in the remaining one-third heart disease was present. He insists on distinguishing the arrhythmia characterized by the short and continuously recurring attacks from ordinary paroxysmal tachycardia, and gives a complete description of its clinical and electrocardiographic peculiarities.

In addition to these references mostly describing repetitive paroxysmal tachycardia separately, a number of examples are included in papers dealing with paroxysmal tachycardia in general. Williams and Ellis (1943) in analysing 36 cases of ventricular tachycardia found 12 belonging to the " intermittent " type. This group which included their only patient with no organic heart disease carried a less serious prognosis than the other group with persistent ventricular tachycardia. In Cooke and White's (1943) list of 32 cases of ventricular paroxysmal tachycardia, Cases 24, 25, and 27, and Freundlich's (1946) Case 3, probably belong to this variety, and so do several of Barker, Wilson, and Wishart's (1943) cases; their Case 1 is our Case 12, and Campbell's (1945) Case 2 is our Case 2.

The various names under which this kind of paroxysmal tachycardia has been described indicate the uncertainty hitherto felt on the subject. While some hold that the relation to exertion or posture is the most characteristic feature, others stress the " incessant," " chronic," or " habitual " nature of the paroxysms. The name " repetitive " seems to us to express best the very frequent recurrence of the paroxysms.

Basis of Investigation

The following account is drawn from a series of 40 patients with repetitive paroxysmal tachycardia. Most of these were seen personally at the Cardiac Department of the London Hospital or at the National Heart Hospital; some were seen privately and others are added through the courtesy of friends. Sometimes the unusual character of the pulse, sometimes the routine cardiogram itself, led to the recognition of this variety of paroxysmal tachycardia. Apart from clinical and electrocardiographic examination, observations were frequent to obtain confirmation of its repetitive nature. The war has made a follow-up more difficult, but some have responded (see Tables).

Clinical Features

Ages extended from the youngest aged 4 years to the oldest aged 75. The auricular form is common in those under 40 and particularly so in children. Flutter and fibrillation in this form are commoner in the adult than in the young, as would be expected. The sex incidence is males : females about 2 : 1.

Observations ranging from 18 years to 3 months was possible in more than half the cases (see case notes and Tables). The longest the paroxysmal state persisted was 10 years. Eleven patients were seen once or twice only. The diagnosis was here based on medical reports of almost incessant tachycardia or arrhythmia, on their long-standing symptoms, and of course on the characteristic electrocardiogram.

Symptoms and clinical features were similar in all groups irrespective of the electrocardiographic pattern. The common symptom was palpitation, usually continuous and independent of exertion; a few complained of slight breathlessness in addition. Six had syncopal attacks, which in three (all ventricular) constituted the main feature (Cases 32, 33, and 39). Unfortunately we never had an opportunity of observing the patient when they occurred. In the absence of evidence of heart-block at any time, we do not regard them as examples of Stokes-Adams attacks (Parkinson, Papp, and Evans, 1941). Symptoms were often present over many years (Table I), and were severe enough in seven (Cases 3, 7, 12, 15, 19, 32, 39) to restrict

TABLE I

CASES OF REPETITIVE AURICULAR PAROXYSMAL TACHYCARDIA

Case No., Sex, and Age	Duration of symptoms when first seen	Under observation	Repetitive paroxysms observed	Follow-up
1. M. 6	1 month	18 years	10 years	Well, no paroxysms.
2. M. 7	—	6 years	5 years	—
3. F. 22	5 years	6 years	6 years	—
4. M. 20	2 years	5 years	5 years	—
5. M. 11	—	7½ years	3 years	Still paroxysms.
6. F. 30	—	3½ years	7 months	—
7. M. 25	2 years	2 years	2 years	—
8. M. 11	—	2½ years	2½ years	—
9. M. 46	—	2½ years	2½ years	Still paroxysms.
10. M. 6	2 years	9 months	9 months	Still paroxysms.
11. F. 11	—	3 months	3 months	Well after 4 yrs. No paroxysms.
12. M. 39	9 years	5 years	6 months	—
13. M. 9	5 years	—	? 9 years	Well after 13 yrs. No paroxysms.
14. M. 59	10 years	4 years	4 years	Still paroxysms.
15. F. 50	20 years	8 months	8 months	Still paroxysms.
16. F. 23	14 months	10 months	3 months	Well, no paroxysms.
17. M. 35	—	5 years	5 years	Still paroxysms.
18. M. 18	—	4 years	4 years	Still paroxysms.
19. F. 20	—	3 years	3 years	—
20. M. 18	—	1 year	1 year	Still paroxysms.
21. M. 18	—	6 months	1 month	Well, no paroxysms.
22. M. 19	—	4 years	2 months	Well, ? paroxysms.
23. M. 39	—	Seen once	—	—
24. M. 37	3 weeks	Seen once	—	—

TABLE II

CASES OF REPETITIVE FLUTTER (CASES 25–28) AND OF AURICULAR FIBRILLATION (CASE 29)

Case No., Sex, and Age	Duration of symptoms when first seen	Under observation	Repetitive paroxysms observed	Follow-up
25. F. 34	months	3 years	3 years	—
26. M. 55	—	Seen once	—	—
27. M. 52	months	Seen once	—	Well after 16 yrs. ? paroxysms.
28. M. 75	5 years	Seen once	—	—
29. M. 56	2 years	Seen once	—	Failure after 4 years.

TABLE III

CASES OF REPETITIVE NODAL PAROXYSMAL TACHYCARDIA

Case No., Sex, and Age	Duration of symptoms when first seen	Under observation	Repetitive paroxysms observed	Follow-up
30. F. 22	3 months	2 years	2 years	Less palpitation.
31. F. 56	13 years	3½ years	3½ years	—

TABLE IV

CASES OF REPETITIVE VENTRICULAR PAROXYSMAL TACHYCARDIA

Case No., Sex, and Age	Duration of symptoms when first seen	Under observation	Repetitive paroxysms observed	Follow-up
32. F. 35	1 year	13 years	2½ years	Well after 12 yrs. No paroxysms.
33. M. 20	4 years	1 year	1 year	Still paroxysms.
34. F. 4	2 months	11 months	11 months	Still paroxysms.
35. M. 36	4 years	4 months	4 months	Well after 10 years. No paroxysms.
36. M. 57	1 year	Seen once	—	Well after 4 yrs. No paroxysms.
37. M. 57	12 years	Seen once (hypertension)	—	Died in failure after one yr.
38. F. 60	1 year	Seen twice	—	Well after 9 yrs. No paroxysms.
39. F. 67	1 year	Seen twice	—	Well after 5 yrs. No paroxysms.
40. F. 45	2 years	1½ months	1½ months	Still paroxysms.

their physical activities. In the others the disability was minimal, though of six who were in the Services, three (Cases 4, 7, 33) had to be discharged. Seven children and seven adults were symptomless.

It is a remarkable fact that evidence of organic heart disease was found only in three patients. Case 37 with the ventricular form had hypertension and cardiac enlargement; he died suddenly three years later in failure. Case 40, also with the ventricular form, had slight cardiac enlargement of obscure origin and inversion of T I. Case 8 with the auricular form had congenital heart disease (atrial septal defect). Case 14 and Case 15, both with the auricular form, had toxic goitre; and Case 32, with the ventricular form for 5 years, had only single ventricular extrasystoles when six years later she was found to have a goitre.

ELECTROCARDIOGRAPHIC FEATURES

The 40 cases were classified according to the electrocardiographic findings as follows:

(1) Repetitive auricular paroxysmal tachycardia, 24 cases.
(2) Repetitive flutter, 5 cases.
(3) Repetitive nodal paroxysmal tachycardia, 2 cases.
(4) Repetitive ventricular paroxysmal tachycardia, 9 cases.

Repetitive auricular fibrillation might have formed another group, but it was seen only once and then only a single cardiogram was obtained. This case is included in Group 2.

(1) REPETITIVE AURICULAR PAROXYSMAL TACHYCARDIA

In 16 patients—they include the six cases of latent and partial block—the same type of extrasystole always formed the runs whether short or long (Fig. 1). P was inverted in leads I and II in one case, in leads II and III in seven, and was upright in six; it was bifid in one case and diphasic in one. If upright, the P always differed in some respect from the normal P of the sinus beat.

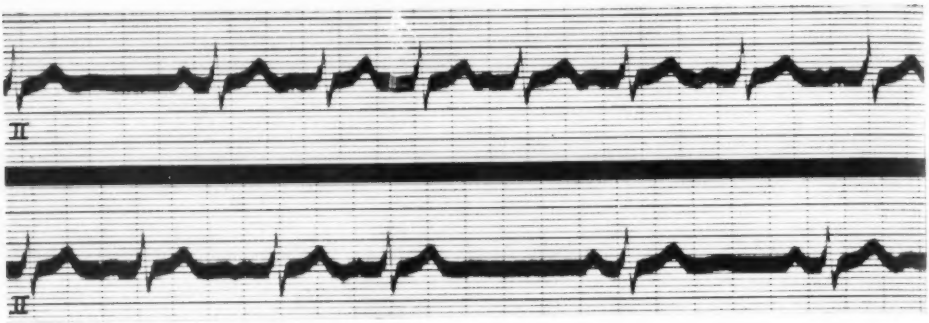


FIG. 1.—Repetitive auricular paroxysmal tachycardia (R.A.P.T.). Case 23. Beginning and end of paroxysm which is irregular, rate 90. Note change in P.

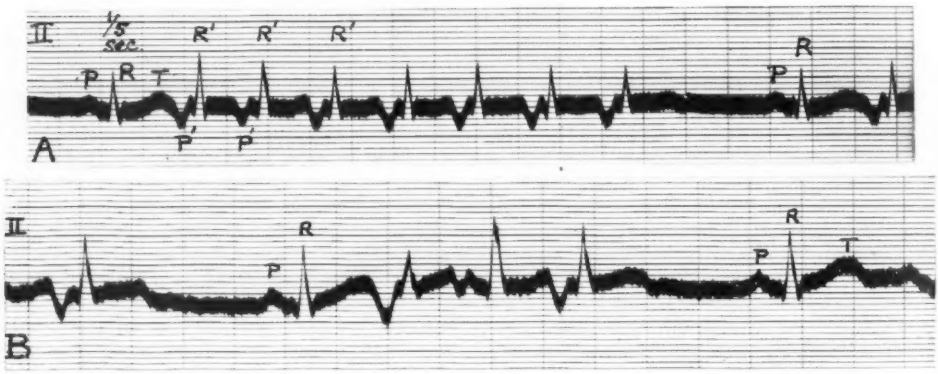


FIG. 2.—R.A.P.T. Case 1. (A) Run of auricular tachycardia, rate 170, regular. (B) Short run of multiform auricular extrasystoles at a similar rate, but irregular.

In five patients, besides the recurrent short paroxysms like ordinary paroxysmal tachycardia as described above (Fig. 2A), there were many other brief paroxysms of an extrasystolic type—apparently runs of multiform auricular extrasystoles, irregular and some with bizarre ventricular complexes (Fig. 2B).

In two patients (Cases 7 and 14) these extrasystolic runs alone were recorded (Fig. 3). In 4 out of 24 cases, blocked auricular extrasystoles added to the disorder of the rhythm (Fig. 4). They were frequent during digitalis treatment. The rate in children was around 150. The rate in adults was noticeably lower; the highest was 150 in Case 3, the lowest 90 in Case 5, and on the average it was 120–130. The rhythm during paroxysms of paroxysmal tachycardia type was regular in 15, more or less irregular in 7 where the paroxysms were of a low rate (around 100) and of a short duration (3–7 beats) (Fig. 1). Even if regular, a slight slowing might occur towards the end of the paroxysm through a slowing of the last beats or through a slight prolongation of the P–R interval.

Repetitive Auricular Paroxysmal Tachycardia with Block. During paroxysms latent block and higher grades of A-V block were found in 7 cases. The P–R interval during paroxysms was prolonged to 0·26 sec. in Case 13; Case 18 had Wenckebach periods (Fig. 5A), and Case 9

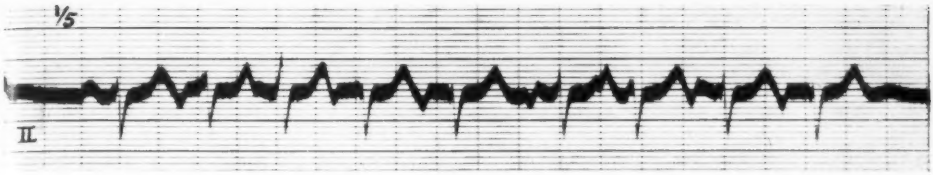


FIG. 3.—R.A.P.T. Case 14. Run of multiform auricular extrasystoles, rate 145, very irregular, forming repetitive paroxysms.

FIG. 4.—R.A.P.T. Case 6. Lead I: run of auricular paroxysmal tachycardia, rate 145, irregular. Lead II: groups of multiform auricular extrasystoles. Lead III: blocked auricular extrasystole.

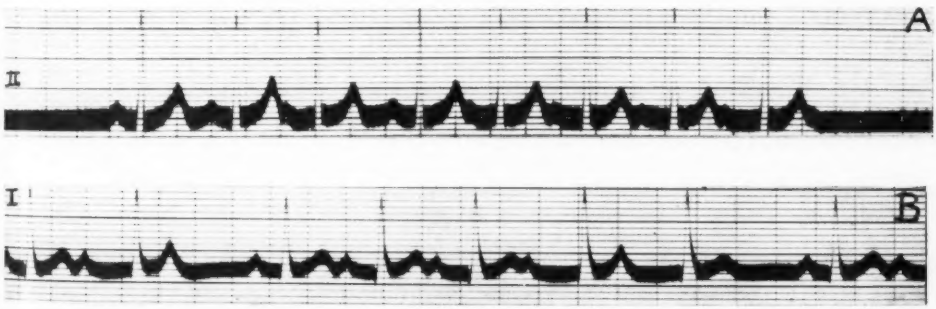


FIG. 5.—R.A.P.T. with Wenckebach periods. (A) Case 18, prolongation of P–R interval at the end of paroxysm from 0·16 to 0·24 sec. (B) Case 9, progressive prolongation of P–R interval from 0·18 to 0·36 sec. during paroxysm. Dropped beats in both cases; rate 120, slightly irregular.

showed at times Wenckebach periods and at other times regular tachycardia at a rate of 130 with P–R 0·2 sec. (Fig. 5B).

Heart block was found in 4 cases. In Case 2, 2 : 1 A-V block developed as soon as the auricular rate rose to about 300, in Case 16 when about 200 (Fig. 6). In Case 12, block of

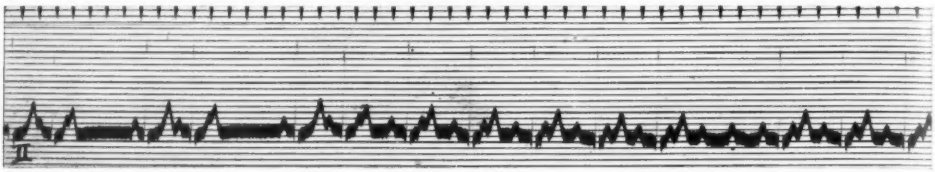


FIG. 6.—R.A.P.T. with 2 : 1 A-V block. Case 16. Auricular rate 200, regular, ventricular rate half.

2 : 1 and of higher degree might be present at a rate of 160–170, while at other times normal conduction was found at a rate of 200. In Case 8, 2 : 1 block was recorded all the time except when quinidine reduced the auricular rate from 180 to 105.

Case 8 had congenital heart disease and he always had a prolonged P–R interval of 0·22; Case 2 when normal rhythm became re-established also had a P–R interval of 0·24 sec. and a right bundle branch block of the wide S type in addition. The P–R interval was also prolonged and P became bifid in Case 12 when seen in sinus rhythm two and five years later. In Case 16, 2 : 1 A-V block was found only on one occasion.

After the observations of Evans (1944) who found 2 : 1 block in 27 consecutive cases of auricular paroxysmal tachycardia, it is remarkable that among our 24 patients with repetitive auricular paroxysmal tachycardia only three had 2 : 1 A-V block. Admittedly the CR_1 chest lead was not applied, but in most of our records (e.g., Fig. 2A) it is obvious that it is a 1 : 1 and not a 2 : 1 rhythm. The slower auricular rate may be a partial explanation.

(2) Repetitive Auricular Flutter

Repetitive flutter is very rare and few examples have been reported. Ritchie (1912) described one as intermittent auricular flutter; Semerau (1918) and Wolferth (1925) each reported two cases of intermittent impure flutter, and East and Bain (1936) one further case (Fig. 135). The physiological mechanism of impure flutter was explained by Lewis (1920) as a variation in the path, or in the speed, of the circus movement from cycle to cycle.

Ordinary flutter in the repetitive form must be exceptional. We observed it in Case 8 with repetitive auricular paroxysmal tachycardia and 2 : 1 A-V block once, and then for a short time only.

In our four cases of repetitive flutter the short paroxysms of 1–3 sec. duration are always introduced by an auricular extrasystole; this is followed by more or less irregular auricular activity at a rate of 200–370 with a completely irregular ventricular activity of 120–200. In Case 28, in whom the paroxysms could be produced at will by swallowing (Fig. 7), there is a waxing and waning of the auricular rate, the approximate distances of the auricular waves during an attack of 8 cycles being 34, 30, 12, 16, 18, 18, 28, 30, and 34 hundredths of a second, corresponding to an auricular rate 180–370. The paroxysms in Cases 26, 27, 28 are followed by a pause, but not in Case 25 (Fig. 8). Case 28 in other records on the same occasion had an almost regular auricular tachycardia (or flutter) with 2 : 1 block.

In the only instance of repetitive auricular fibrillation (Fig. 9) the paroxysm in lead II is introduced by an auricular extrasystole, followed by another which initiates auricular fibrillation. Lead I of the same record shows auricular fibrillation; lead III sinus rhythm with auricular extrasystoles. The history favoured the diagnosis of repetitive auricular fibrillation.

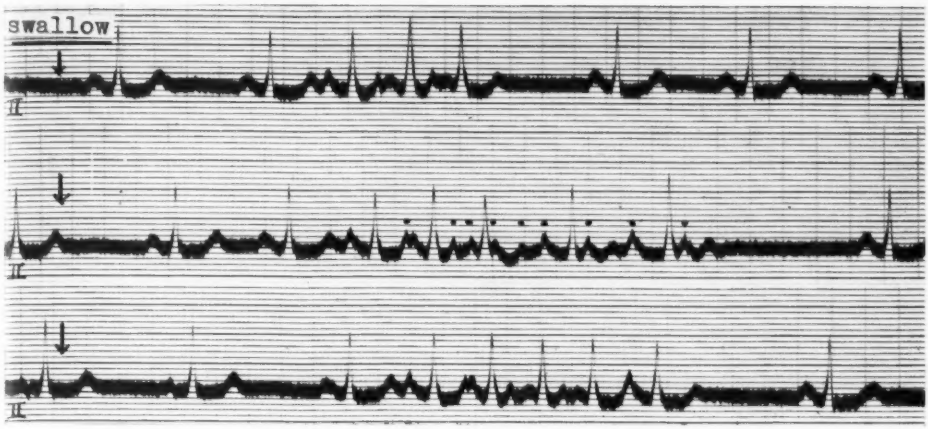


FIG. 7.—Repetitive flutter; each paroxysm on swallowing. Case 28. Waxing and waning of auricular rate from 340 to 200 (dotted), ventricle irregular 200–100.

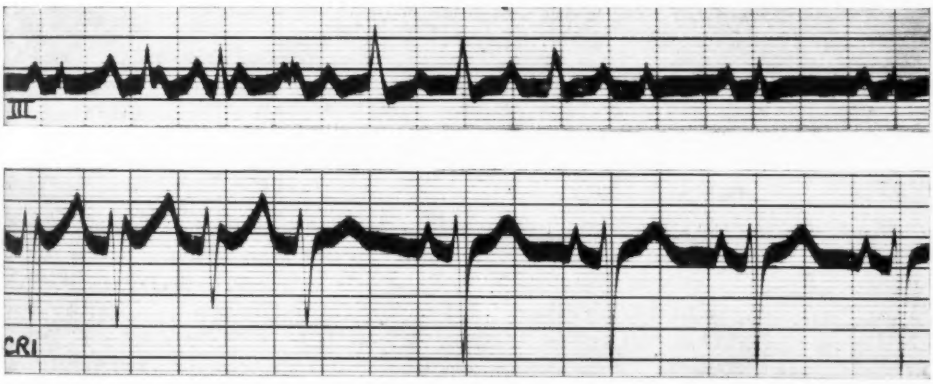


FIG. 8.—Repetitive flutter. Case 25. Auricular rate 380, slightly irregular; ventricular rate 160, irregular. CR_1 chest lead: flutter waves.

but the only record obtained of it is that here described. We have since seen another case of this kind with a similar electrocardiogram.

(3) Repetitive Nodal Paroxysmal Tachycardia

Though Case 1 was once seen in inferior nodal tachycardia, and isolated nodal extrasystoles occasionally appear in records of repetitive auricular paroxysmal tachycardia, repetitive nodal paroxysmal tachycardia itself is rare and only two examples were found.

Case 30 shows regular nodal rhythm at a rate of 64, with occasional sinus beats while at rest. Sinus rhythm at rest was recorded only once during two years of observation. Moderate effort produced nodal tachycardia at a rate of 130–140 with no visible P waves. In Case 31 an inverted P follows R at a short distance, and gradual slowing occurs at the end of paroxysms (Fig. 10).

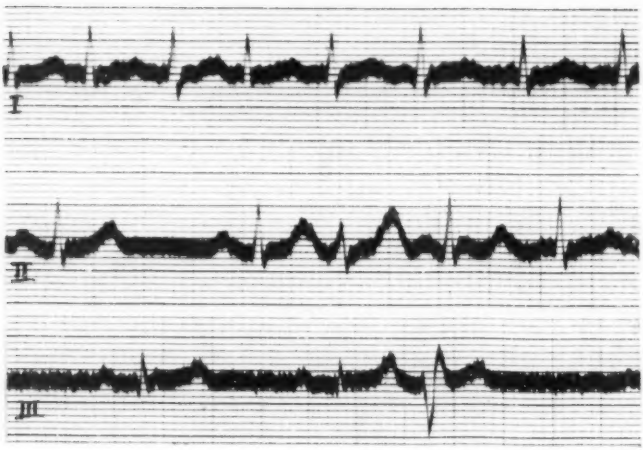


FIG. 9.—Repetitive auricular fibrillation. Case 29. Lead I: auricular fibrillation. Lead II: auricular extrasystole initiating period of fibrillation. Lead III: normal rhythm with auricular extrasystoles.

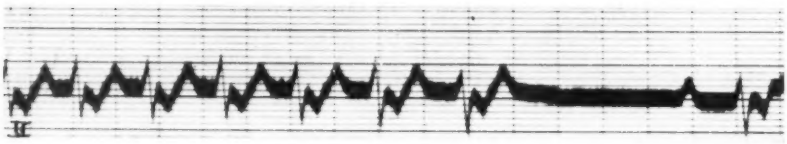
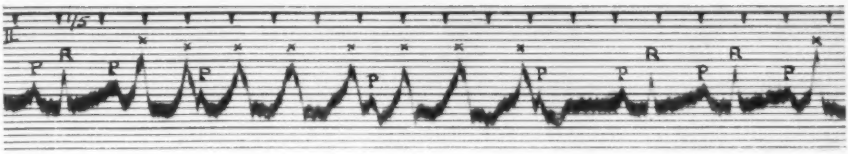


FIG. 10.—Repetitive nodal paroxysmal tachycardia. Case 31. End and beginning of paroxysm. P–R interval, 0·24 sec. after paroxysm.

(4) Repetitive Ventricular Paroxysmal Tachycardia

Case 32 sometimes showed ventricular paroxysmal tachycardia with uniform ventricular complexes at an approximate rate of 200 and an auricular rate of 150 (Fig. 11A), and sometimes multiform ventricular extrasystoles in groups separated from each other by one or two sinus beats (Fig. 11B). Case 33 had regular ventricular tachycardia at a rate of 120 when seen twice in 1943 and once in 1944. Under digitalis treatment sinus rhythm reappeared for a short time; a few days later, however, in spite of continued digitalis treatment, runs of ventricular extrasystoles were again found, and after one month these repeated runs were still present (Fig. 12). Extrasystoles of different origin can be seen in the records of Case 39, but runs of ventricular tachycardia are always produced by the same interpolated ventricular extrasystole and are not followed by a pause (Fig. 13A and B). Case 38 (Fig. 14) with incessant bouts of irregular ventricular tachycardia at a maximum rate of 260, the highest in the series, was well and free from attacks when seen nine years later at the age of 69. The irregularity is even more obvious in Case 40 (Fig. 15) where retrograde conduction is evident as it is in Case 33 (Fig. 12) and Case 34. Except for Case 37, who died three years later, Case 40 is the only one with an enlarged heart and an abnormal cardiogram which here showed an inversion of T I of the sinus beat (Fig. 15). In all other records of this series the intervening sinus beats were normal and even during prolonged repetitive paroxysmal tachycardia the T of the sinus beats never seemed to change from upright to inverted. This is surprising in



A

B

FIG. 11.—Repetitive ventricular paroxysmal tachycardia (R.V.P.T.). Case 3. (A) Run of ventricular tachycardia, rate 240, almost regular; auricular rate 150. (B) Run of multiform ventricular extrasystoles at a similar rate, but irregular.

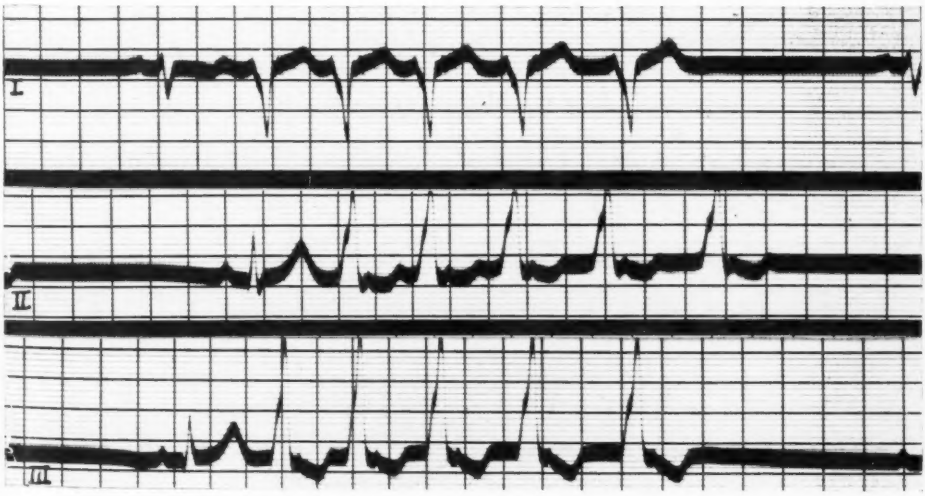


FIG. 12.—R.V.P.T. Case 33. Gradual waning of ventricular rate from 140 to 110. Retrograde auricular conduction seen in leads II and III.

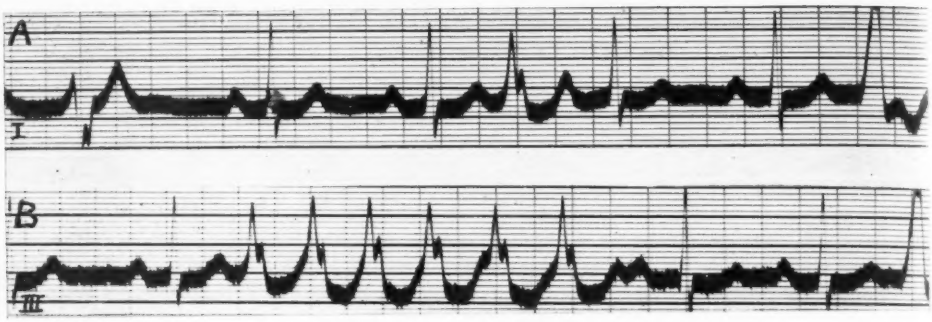


FIG. 13.—R.V.P.T. Case 39. (A) Multiform ventricular extrasystoles, one interpolated, identical with those in B. (B) An interpolated paroxysm at a regular rate of 200. P–R interval prolonged to 0·26 sec. after paroxysms.

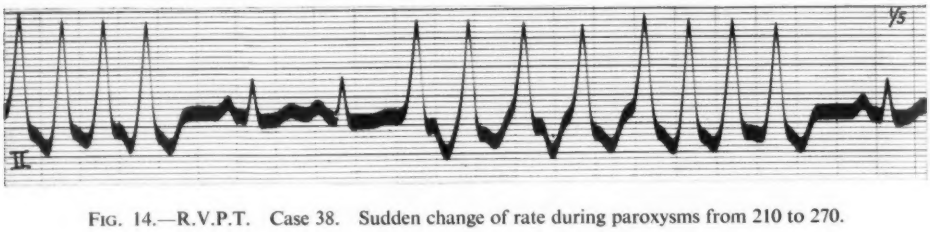


FIG. 14.—R.V.P.T. Case 38. Sudden change of rate during paroxysms from 210 to 270.

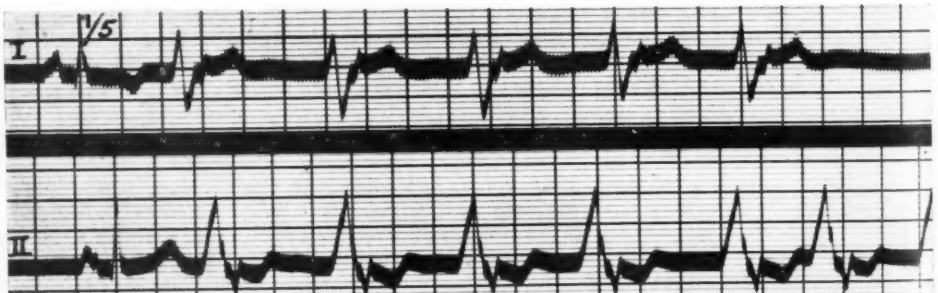


FIG. 15.—R.V.P.T. Case 40. Rate 80–150: irregular. Retrograde auricular conduction. T I inversion of sinus beat.

view of the frequency with which T inversion during normal rhythm following prolonged paroxysmal tachycardia has been reported.

The Nature of the Condition

Although the single beats of ordinary paroxysmal tachycardia closely resemble those of extrasystoles, the relation between the two is not as close as it may seem. Thus, extrasystoles are the most frequent of arrhythmias while paroxysmal tachycardia is uncommon. Further, the majority of patients with ordinary paroxysmal tachycardia do not show extrasystoles in their normal rhythm. Campbell and Elliott (1939) found them in only 16 cases of their 42 cases with recorded paroxysms, and then they were often of a type different from the beats constituting the paroxysm. Isolated ventricular extrasystoles were found between attacks both of supraventricular and ventricular tachycardias.

In repetitive paroxysmal tachycardia short spells of extrasystoles, varied in form and irregular in rhythm, often recur constituting tachycardiac runs. These, however, are not the only constituents in repetitive paroxysmal tachycardia; even more often there are typical short paroxysms of tachycardia with all the beats similar in form and exactly like ordinary paroxysmal tachycardia except in their brevity and constant recurrence. The rhythm is mostly regular, though during brief and low-rate paroxysms arrhythmia (? sinus) may be found (cf. " nomotopic tachycardia," Maddox, 1937). Many years after the repetitive paroxysmal tachycardia has ceased, frequent single extrasystoles are still often to be found. As we see it, repetitive paroxysmal tachycardia provides a connecting link between extrasystoles and ordinary paroxysmal tachycardia, because it is composed both of runs and salvos of extrasystoles and of short ordinary paroxysms. Lewis (1909) expressed the same view about his relevant case of paroxysmal tachycardia already cited, and Katz (1946) illustrates the common genesis of extrasystoles and paroxysmal tachycardia in published records (Fig. 385, 386, 391, 395, and 396). Similar links can be established between auricular extrasystoles —e.g., repetitive auricular tachycardia—and auricular flutter and fibrillation. In Case 20 short periods of flutter are mixed with the repetitive runs of auricular tachycardia; in Case 28 auricular paroxysmal tachycardia was recorded at a time when the patient exhibited repetitive impure flutter, and the reverse occurred in Case 8 with repetitive auricular tachycardia and 2 : 1 A-V block who once showed pure flutter. Case 14 with repetitive showers of multiple auricular extrasystoles was twice recorded in auricular fibrillation. Repetitive flutter was always introduced by an auricular extrasystole, and so was repetitive auricular fibrillation in our only case. Repetitive auricular tachycardia illustrates better than any other condition the interdependence of arrhythmias. The relationship between the single groups may at times be so close that electrocardiographic distinction may be impossible—as it often is between auricular paroxysmal tachycardia with 2 : 1 A-V block and flutter (Evans, 1944).

Ætiology. Repetitive paroxysmal tachycardia apparently is not due to inflammatory, degenerative, or toxic causes. Though two of our patients gave a history of rheumatic fever or chorea, none had rheumatic heart disease; and the infections preceding the arrhythmia in a few, e.g., diphtheria in Case 31, glandular fever in Case 7, may have been incidental. Only one of the nine with repetitive ventricular tachycardia had hypertensive heart disease, and none of them had angina pectoris in any form. Heavy smokers and drinkers were scarce in our series. An objection to organic heart disease as a cause is that the basic cardiogram between the paroxysms and after an interval of years, with two exceptions always remained normal. In three cases an abnormal thyroid may have been the source of this arrhythmia. Case 32 was found to have a goitre with slight hyperthyroidism when seen five years after she had been under observation for repetitive ventricular paroxysmal tachycardia. Case 15, with the auricular form, had an enlarged thyroid. Case 14 had a slight thyroid enlargement and a fine tremor, and mild hyperthyroidism was diagnosed by us and the next year by another

observer who proposed a subtotal thyroidectomy which was refused: the next year paroxysmal fibrillation appeared in addition to repetitive paroxysmal auricular tachycardia as before.

The relative rarity of paroxysmal tachycardia of any type, its occurrence in infancy, childhood, and early adult life, its occasional association with congenital heart disease (Case 8), in default of any other convincing cause at least permits us to suggest that it may be due to a congenital peculiarity in the conducting (specific) system of the heart. A recent case of congenital paroxysmal tachycardia diagnosed during fœtal life and confirmed after birth is described by Garvin and Kline (1947). Congenital heart block is already fully accepted. The current opinion about the short P–R bundle branch block condition (the so-called W.P.W. syndrome) is that it depends upon a congenital anomaly in the conducting system, and if so the high incidence of paroxysmal tachycardia in that syndrome points to a common congenital ætiology for both. That is also the view of Fine and Miller (1940). Admittedly the disturbance may arise in the nervous control of the heart and possibly from a congenital anomaly there. The relation to emotion or exertion (Cases 19, 30), and in one particular instance to swallowing (Case 28) (Fig. 7), intimates that the mechanism is at least under nervous control.

Diagnosis. When first clinically examined a patient was often thought to have auricular fibrillation or flutter, though sometimes the unusual nature of the case was suspected from the pulse. In general the condition cannot be recognized clinically; and the diagnosis rests upon the cardiographic recording of constantly recurring short paroxysms of tachycardia separated from each other by single or several sinus beats, sometimes more. This paroxysmal state continues for weeks or months or even years. In a proportion including some of the ventricular variety, there are also numerous short runs or salvos of multiform extrasystoles, irregular in rhythm. Contrary to the opinion of Froment (1932) we think that the cardiogram of such cases (Fig. 11B) in no way differs from that of " grave " or " terminal " ventricular tachycardias except that normal sinus beats are also in evidence. Sometimes short extrasystolic runs alone constitute the repetitive brief paroxysms.

Certain allied conditions will be discussed shortly.

(1) *Persistent paroxysmal tachycardia* (or ectopic auricular tachycardia) seems to be interminable and every record shows nothing but " paroxysmal tachycardia " for months and years. Such has been described by Weiss and McGuire (1936). Of this condition we have records of two cases, both children, one (P. T.) aged $2\frac{1}{2}$, and the other (R. W.) aged 6 years. In the first the " paroxysm " seems to have persisted for almost three years at a regular rate of 170 and the cardiogram has always shown inverted P I, bifid P II, and diphasic P III; in the second with a similar observation period and rate there was always a diphasic P I with inverted P II and P III. Prolonged observation in hospital was possible in the second child,

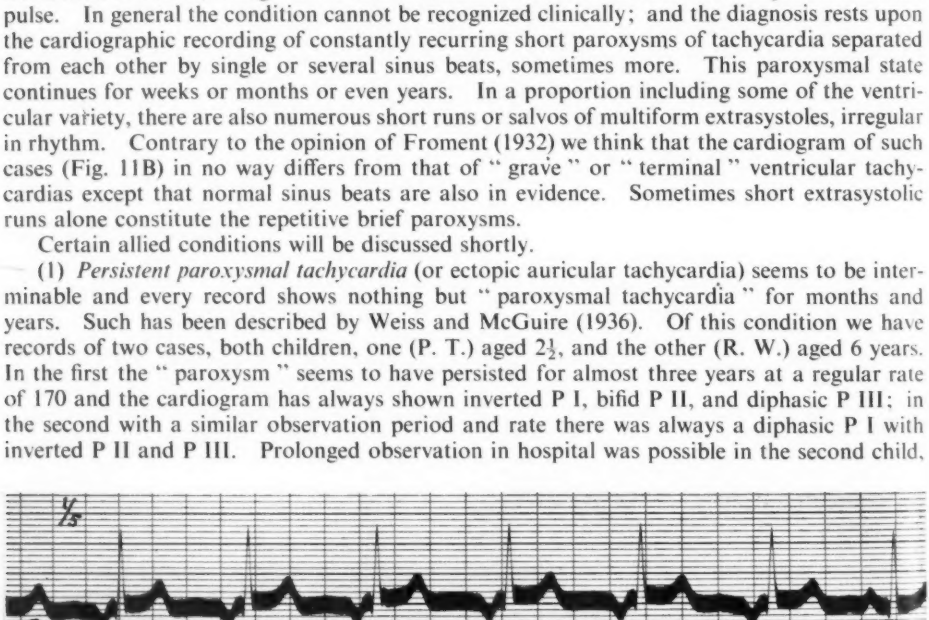


FIG. 16.—" Auricular rhythm." Case G. F. Change in rhythm: inversion of P and slight shortening of P–R interval—without change in rate.

and under digitalis treatment sinus beats and transient sinus rhythm appeared, but the ectopic tachycardia recurred as soon as the effect of the drug wore off. Herson and Willington (1947) made a similar observation. In Cases 11, 15, and 33, digitalis broke up the apparently persistent paroxysms into repetitive paroxysmal runs which persisted after treatment was stopped. Thus the difference between persistent and repetitive paroxysmal tachycardia, based on the frequent appearance of sinus beats in the latter, is not great because persistent paroxysmal tachycardia under hospital observation and digitalis treatment can often be transformed, at least temporarily, into the repetitive variety.

It is worthy of note that runs of irregular and multiform extrasystoles always seem to be short and do not in nature compose the long attacks that characterize ordinary paroxysmal tachycardia as distinct from repetitive paroxysmal tachycardia.

(2) *Auricular rhythm without tachycardia.* The term auricular rhythm is here used in contrast with sinus rhythm, and it is comparable with nodal rhythm. It comprises instances in which the P of the sinus beat suddenly changes to an abnormal P without any increase in rate. This condition, described by Scherf and Harris (1946) as "coronary sinus rhythm," was observed in three cases, not included in our series for there was no tachycardia during the abnormal rhythm marked by the sudden inversion of P I and P II (Fig. 16). The close relationship of auricular rhythm to repetitive paroxysmal tachycardia is shown by our Case 12 (vide Fig. 1 (C) in Barker *et al.* 1943), and the one of Maddox (1937); in both of them an ectopic P identical with the one during paroxysms continued when the paroxysms were over and the rate was slow. Normal sinus rhythm was not seen.

(3) *Sinus arrhythmia.* The short and low-rate irregular paroxysms of repetitive auricular paroxysmal tachycardia may be mistaken for sinus arrhythmia. Distinctive features are the ectopic P waves of all but the first complexes of the runs, and the pause that follows the runs (Fig. 1).

Course, Treatment, and Prognosis

The case notes (see appendix) will illustrate the course of repetitive paroxysmal tachycardia. Children often grow out of it at the time of adolescence. The state of paroxysms subsided in Cases 1 and 13 at the age of 14 and 15 years, and Case 11, observed in a state of paroxysms for three months when 11, was found in normal rhythm four years later. Case 5 forms an exception; observed for sinus tachycardia since the age of 11, he had repetitive auricular paroxysmal tachycardia when 15 and still had it when last seen at the age of 18. Among 14 adults who could be followed up, six (Cases 9, 14, 17, 18, 20, and 33) had repetitive paroxysmal tachycardia one, four, and five years after it was first detected; while eight had either a normal cardiogram when re-examined or had stated they were free from palpitation when questioned by letter. Among them were Cases 32, 35, 36, and 38 with repetitive ventricular tachycardia in whom the state of paroxysms was known to have lasted from 2½ years (Case 32) to 4 months (Case 35). The state of paroxysms in most patients did not cease abruptly, but the paroxysms gradually became less frequent, then occurred at long intervals, eventually perhaps disappearing altogether.

The disability arising from repetitive paroxysmal tachycardia differs according to the patient, and the rate and duration of the paroxysms. Children tolerate almost continuous tachycardia at a rate of 160–180 surprisingly well, being generally symptomless; and so do adults if not subjected to excessive physical strain. Case 15 sustained successfully the operation of bilateral salpingo-oöphorectomy complicated by postoperative bronchopneumonia, while in the paroxysmal state with a pulse rate of 120–160.

The course and duration of repetitive paroxysmal tachycardia cannot be much modified by treatment, though the paroxysms disappeared in Case 6 after two months' quinidine. An almost continuous ventricular tachycardia in Case 33 and a continuous auricular tachycardia in Cases 11 and 15 were transformed into a repetitive one by digitalis. Unsuccessful attempts

with both digitalis and quinidine were far more numerous (Cases 1, 3, 4, 13, 7, and 12). Nor had vagal compression and deep breathing any effect when tried during prolonged paroxysms.

The prognosis in general is certainly better than one might at first suppose. Though therapeutic attempts usually fail there is good reason to expect that in time the state of recurrent tachycardia will pass. Meanwhile most patients are able to live an almost normal life provided they avoid great physical activity. Our series of prolonged observations includes only three cases where heart disease was already evident; of these, two were little affected but one of them (Case 37) was mortally aggravated by repetitive tachycardia—ventricular in this case.

Summary

The name " repetitive " is here applied to a special variety of paroxysmal tachycardia in which short paroxysms separated by sinus beats constantly recur over months or years.

Forty new cases of repetitive paroxysmal tachycardia have been collected, nineteen of them with prolonged observation periods ranging from two months to eighteen years, and their clinical and electrocardiographic features were studied. Ages extended from 4 to 75 years, with two-thirds of the patients (including seven children) under 40. Males were affected twice as often as females.

The usual symptom was palpitation, and four patients with the ventricular form had syncopal attacks in addition. With few exceptions the disability caused by the recurrent paroxysms was surprisingly slight. Fourteen patients—seven children and seven adults—had no symptoms, and all but one of these had repetitive auricular tachycardia.

Of the forty cases only three had evidence of organic heart disease. One case with the ventricular form had hypertension and cardiac enlargement, another had cardiac enlargement of unknown origin, the other with the auricular form had congenital heart disease. Toxic goitre was found in two with the auricular form and in one with the ventricular form. Two gave a rheumatic history but had no rheumatic heart disease.

An irregular pulse was the only clinical sign. The diagnosis was based on finding in a patient at numerous consecutive examinations a particular kind of electrocardiogram. This showed on almost every occasion, short paroxysms of tachycardia composed of several beats or more, and separated by normal sinus beats. The electrocardiogram in its rhythm was divided as follows.

(*a*) Repetitive auricular paroxysmal tachycardia, 24 cases. In 16 there were short runs of the ordinary pattern; in 5 cases there were in addition salvos of multiform auricular extrasystoles which in 3 of these formed alone the brief irregular paroxysms. The rate was lower than in ordinary paroxysmal tachycardia, being on the average 150 in children and 130 in adults.

(*b*) Repetitive auricular flutter, 5 cases. This differed from ordinary flutter in that the auricular rhythm was irregular (impure flutter). One here included was probably fibrillation.

(*c*) Repetitive nodal paroxysmal tachycardia, 2 cases.

(*d*) Repetitive ventricular paroxysmal tachycardia, 9 cases, in brief, repeated paroxysms, identical with ordinary ventricular tachycardia (including the grave variety) in respect of the electrocardiogram, except that the sinus beats between paroxysms were physiological, giving no evidence of organic heart disease.

Repetitive paroxysmal tachycardia provides a connecting link between extrasystole and paroxysmal tachycardia because it may be composed both of runs of extrasystoles and of short ordinary paroxysms. The extrasystolic type is irregular and the paroxysmal tachycardia type is more often irregular than is ordinary paroxysmal tachycardia. The ætiology of repetitive paroxysmal tachycardia is unknown though something can be said for the view that it arises from a congenital anomaly of the conducting system.

The paroxysmal state is uncertain in its duration, but it often subsides. In children it

may last until adolescence; in adults it may last for years and then disappear as it did in 8 out of 14 cases long observed. The prognosis as regards disability and length of life is good both in the auricular and the ventricular form; naturally there are exceptions. Quinidine may be tried especially in the ventricular form, but the results are meagre; digitalis is also disappointing; neither is likely to terminate the state of paroxysms. Repetitive paroxysmal tachycardia may be regarded as a distinctive disorder of rhythm rather than a cardiac disease of consequence.

Illustrative Cases

List of Abbreviations

A.Ex. = auricular extrasystole(s).
A.P.T. = auricular paroxysmal tachycardia.
B.B.Bl. = bundle branch block.
B.P. = blood pressure.
EC. = electrocardiogram(s).
N.R. = normal rhythm (sinus rhythm).
R.A.P.T. = repetitive auricular paroxysmal tachycardia.
R.V.P.T. = repetitive ventricular paroxysmal tachycardia.
V.Ex. = ventricular extrasystole(s).
V.T. = ventricular tachycardia.

Case 1, *aged* 6.

Repetitive tachycardia in a boy aged 6, *almost continuously present for ten years without symptoms or signs other than a rapid irregular pulse. Electrocardiogram* (*EC.*) *almost always showed runs of regular auricular tachycardia interspersed with sinus beats; also groups of multiform auricular extrasystoles. Digitalis and quinidine unsuccessful. Normal school life, then gradual and spontaneous disappearance of the arrhythmia after the age of* 16. *Well and at work, free from paroxysms at the age of* 27 (21 *years' observation*).

History. He was a twin and weighed only 4½ lb. at birth. He had always been fit until the last few weeks when his mother noticed that the heart was beating fast, and his father, a doctor, found the pulse to be uncountable. The child did not complain and had always been particularly active.

Examination (1925). Well-developed, of good colour, not distressed. No abnormal signs except for irregular tachycardia varying from 120–180. Heart sounds clear; no cardiac enlargement on radioscopy. EC. (Fig. 2): short runs of A.P.T. and multiple A.Ex. interspersed with occasional normal beats.

Course. From 1925 till 1943 he was examined at frequent intervals; he was also under close observation during the first five years by his father, who for one year charted the pulse three times a day. The child had hardly a day without paroxysms or very frequent extrasystoles, so that it was uncommon to find the pulse completely regular; neither absolute rest in bed nor moderate exertion made any difference, and during sleep at times the pulse was found as irregular. Quinidine had no effect when given for two months, nor had digitalis when given for three weeks, both in moderate doses. Climatic change was without effect, for the pulse was as irregular in the Highlands as it was at the seaside. Intercurrent diseases left the paroxysms unaffected: in 1926 during a period of acidosis with renal glycosuria, pyrexia, and vomiting, the paroxysms were as before. From 1929 onwards he lived an almost normal life for a schoolboy of his age. He took part in light games, drill, and physical exercises, first at public school, later at the university, avoiding only hard games and competitive sports. In 1932, when 13, the paroxysms became less frequent, and when seen in 1933 there were no extrasystoles or paroxysms. He still had occasional palpitation, and in 1935 runs of paroxysmal tachycardia were again recorded. The EC. was normal during the last two visits in 1939 and 1943 when he was working as a curate, and only occasionally had rapid and irregular heart action. EC. were recorded first at monthly, later at 3–6 monthly periods till 1932, once in 1933, twice in 1935, and again in 1939 and 1943. Outside the paroxysms they are remarkable for the constant pattern all

through the 18 years of recorded observation and except for Q III are normal. During the paroxysmal attacks either runs of multiform A.Ex., mostly 3–5 in number, follow each other at irregular intervals at a rate of 160–180, or longer stretches of regular A.P.T. composed of the same elements persist at a rate of 140–150. These two features can be found side by side on the same record (Fig. 2). Once a short paroxysm of nodal tachycardia was recorded. In January 1947 he was reported to be well and at work, and he had no recurrence of palpitation; this was at the end of 21 years' observation.

Case 2, *aged* 7.

Arrhythmia found on routine examination in a boy of 7, *constantly present till the age of* 12. *No complaints and no organic heart disease. EC. almost always showed repetitive auricular paroxysmal tachycardia with* 2 : 1 *A-V block and an auricular rate of* 300. *After* 5 *years, spontaneous reappearance and persistence of sinus rhythm with P–R interval of* 0·24 *and right bundle branch block in the EC.*

History. Tonsils and adenoids removed 8 months ago. No complaints, energetic, not short of breath.

Examination (October 1930). Short systolic murmur at the apex. No cardiac enlargement. Pulse completely irregular, at first regarded as extreme sinus arrhythmia. EC. short and long runs of A.P.T. interspersed with periods of normal rhythm. During A.P.T. the ventricular complex is modified and resembles that of right bundle branch block; closer inspection shows the deep S in lead II to be due to an inverted P and thus during paroxysm 2 : 1 A-V block persists with an auricular rate of approx. 300 and a ventricular rate of 150.

Course. Under observation (Dr. Maurice Campbell) in Guy's Hospital in November and December 1930, because of otitis media. EC. were always the same, a basic rate of 90–110 suddenly interrupted by a run of A.P.T. at a ventricular rate of 140–170. Effort and deep breathing had no effect on the paroxysms. Seen at frequent intervals from 1930–5; transient sinus rhythm was recorded only twice, the next record on the same day again showed A.P.T. In 1936 sinus rhythm seemed to be re-established at a rate of 75; P–R of 0·22 sec. and right bundle branch block (deep S type) persisted in the EC. He felt well and did not complain.

Case 3, *aged* 22.

Young woman complaining of attacks of faintness, actual faints, and palpitation for five years. Normal heart except for irregular tachycardia. EC. showed repetitive auricular paroxysmal tachycardia. During five years of observation only twice was transient sinus rhythm recorded. All treatment failed.

History. Five years ago " fainting " attacks rarely producing unconsciousness; in hospital for 3 weeks. For 3 years frequent recurrence of faint feelings accompanied by palpitation; also slightly breathless on exertion.

Examination (March 1931). Varying, irregular pulse, 80–110. B.P. 125/70. Clear heart sounds; radioscopy normal. EC. sudden runs of slightly irregular A.P.T. at a rate of 130–140, preceded by a normal beat and followed by a pause. P II and P III inverted.

Course. Heart Hospital in-patient from March till July 1931. During this period the pulse was hardly ever found to be regular, though the rate occasionally dropped to 70. Usual rate at rest 120–140. Quinidine, quinine, digitalis, and atropine had no effect on the rhythm. EC. were almost always of R.A.P.T.; when the paroxysms were short the rate was irregular because of slowing towards the end, when they were long it was regular, 130. Normal rhythm was recorded only three times, at a rate of 80–100. Seen again in February 1932 because of fainting in the street while having palpitation; EC. was found unchanged except for a prolongation of the P–R interval to 0·22 sec. towards the end of the paroxysms. Under observation as out-patient from April 1935 till January 1936 still complaining of giddiness and faintings; heart and B.P. were again found normal, and EC. showed R.A.P.T. on every occasion.

Case 4, *aged* 20.

Man, aged 20, *discharged from the Army for D.A.H. complaining of palpitation for many years. Normal heart except for arrhythmia, rate* 120. *EC. showed repetitive runs of auricular paroxysmal tachycardia. During* 5 *years of observation not once seen in sinus rhythm. Quinidine ineffective.*

History. Discharged from the Army in 1918 because of disordered action of the heart. Complains of continuous palpitation, breathlessness, and slight cough, but at work.

Examination (April 1920). Pulse 130, regular. Heart sounds normal. No cardiac enlargement. EC., A.P.T., 132, regular, P II and P III inverted.

Course. Observed as an out-patient till February 1925. EC. taken first at monthly then at three-monthly intervals always showed either continuous, regular A.P.T. at a rate of 120–130, or short runs of the same pattern preceded by a normal beat and followed by a pause of 1·2 to 1·4 sec. Occasional normal beats were followed by a blocked A.Ex.; at one time single V.Ex. were seen. Quinidine was ineffective.

Case 6, *aged* 30.

Woman, aged 30, *complaining of palpitation. No heart disease. EC. during* 9 *months' observation always showed repetitive runs of auricular paroxysmal tachycardia and groups of multiform auricular extrasystoles. Disappearance of paroxysms under quinidine.*

History. Frequent attacks of sudden palpitation.

Examination (December 1938). No heart disease. Radioscopy normal. EC. runs of nodal and auricular Ex., some with aberrant ventricular complex; at times periods of irregular ventricular activity at a rate of 170 with no visible P waves (? fibrillation). Paroxysm often begins with a nodal beat and is always followed by a pause (Fig. 4).

Course. Observed as an out-patient till August 1939 at monthly intervals, from then till June 1942 at six-monthly intervals. EC. always similar, with shorter or longer runs of Ex. and A.P.T. till May 1939. Quinidine was tried and when seen in July she had only single A.Ex.; quinidine 3 grains t.d.s. continued for three months; from August 1939 onwards, she was only seen in sinus rhythm, though still having occasional palpitation.

Case 8, *aged* 11.

Boy of 11 *with congenital heart disease, probably atrial septal defect, and a rapid irregular pulse. EC. repetitive auricular paroxysmal tachycardia with* 2 : 1 *A-V block, auricular rate* 200. *Persistence during* $2\frac{1}{2}$ *years of observation; once in flutter, never in sinus rhythm. Quinidine reduced auricular rate, otherwise ineffective.*

History. Delicate from birth; as a baby had blue lips, and as a child was unable to play games because of shortness of breath. When aged 5, school doctor sent him to special school because of heart disease.

Examination (February 1924). High colour and slight cyanosis of lips; no dyspnœa. Pulse 100, very irregular, with frequent long pauses. Soft systolic murmur loudest at the pulmonary area and over 3rd and 4th left spaces; no thrill. Pulmonary second sound heard. Radioscopy: prominence and increased pulsation of pulmonary artery with large hilar branches, right ventricle enlarged. EC. runs of A.P.T. with P II and P III inversion; 2 : 1 A-V block. Auricular rate 180–220 irregular, ventricular rate half. Very occasional aberrant beats.

Diagnosis. Congenital heart disease, probably atrial septal defect.

Course. Out-patient (National Heart Hospital) from February till December 1924, seen at frequent intervals; EC. always the same. In-patient from January 31st till March 14th, 1925. During this period of close observation not once was sinus rhythm recorded and the paroxysmal state persisted all the time. The auricular rate was usually 190 with 2 : 1 block; only occasionally were higher grades of block recorded. The short paroxysms usually ended with a pause of 1·2 sec. or less, succeeded by a sinus beat and then a new run. The P–R interval of the sinus and paroxysmal beats was identical and slightly prolonged to 0·22 sec. Once a paroxysm of flutter was recorded, the auricular rate being 308, the ventricular half this and quite regular.

Vagal pressure had no effect on the EC. *Exercise*, paroxysms became shorter, otherwise no effect. Atropine, 1/100 of a grain subcutaneously, increased average auricular rate from 190 to 220. Quinidine reduced the auricular rate of 105 and abolished 2 : 1 block, but only temporarily.

From January till October 1926 seen frequently as an out-patient; the same abnormal rhythm was recorded on every occasion.

Case 14, *aged* 59.

Man, aged 59 *complaining of irregular heart beating. Pulse irregular; thyroid enlarged with mild toxic signs; no cardiac enlargement. During* $3\frac{1}{2}$ *years of observation EC. almost constantly showed runs*

of multiform auricular extrasystoles at a rate of 140–160 *and irregular. Sinus rhythm was exceptional; transient auricular fibrillation twice recorded. Digitalis ineffective.*

History. B.P. found raised 10 years ago when for the first time felt " fluttering and quivering " in the chest at night. For three months missed beats, coupling, or else a completely irregular heart. Feels fit and walks any distance.

Examination (October 1942). Pulse irregular, 70–80. B.P. 160/95. Normal heart sounds; no cardiac enlargement. Isthmus of thyroid gland easily seen; fine tremor of hands; toxic goitre suspected. EC. (Fig. 3) runs of A.Ex. (8–10) of different origin at an irregular rate 140–160, followed by pause. Basic rate, 66 regular.

Course. June 1942 observed at Liverpool Royal Infirmary (Dr. Wallace-Jones). EC. invariably the same. B.M.R.+27 per cent. Iodine treatment begun; reported August 1943, less palpitation under iodine, but EC. unchanged. Subtotal thyroidectomy considered but not performed. From May 5th till June 9th, 1944, in Manchester Royal Infirmary (Prof. Crighton Bramwell); diagnosis of toxic goitre doubted. B.M.R.+5 per cent. Among 28 EC. there recorded, 23 show runs of multiform A.Ex. sometimes interspersed with sinus beats, 3 sinus rhythm, and 2 auricular fibrillation. This last appeared under digitalis which was ineffective in reducing the paroxysms but increased the number of blocked A.Ex.

Seen again in July 1944, February and September 1945, and March 1946; paroxysms recorded on each occasion.

Case 18, *aged* 18.

Healthy and active man of 18, *no complaints. Referred by Medical Board because of irregular pulse. This found at the age of* 14 *on routine examination. EC. then as now, showed repetitive auricular paroxysmal tachycardia with Wenckebach periods.*

History. Played games at school. On admission as naval cadet, aged 14, doctor noticed irregular pulse. EC. (inspected) then showed short runs of A.P.T. similar to the one described below. Completed full physical training of cadet, and never complained of palpitation. Referred by Medical Board because of irregular pulse.

Examination (February 1947). Pulse very irregular with series of rapid beats then pause; persistent, no change on exertion. B.P. 130/80. Normal heart sounds. X-rays, no enlargement EC. continuous short runs (3–7 beats) of A.P.T. at a slightly irregular rate of 120–130 with gradual increase of P–R interval from 0·18 to 0·24 (Fig. 5A). The first two P waves of the paroxysms appear normal, and are small and upright; then P changes, often gradually, to flat-diphasic (lead II). The paroxysm sometimes ended with a blocked P wave, always followed by a pause of varied length.

Case 25, *aged* 34.

Woman, aged 34, *complaining of palpitation. Irregular pulse found with short paroxysms of tachycardia; otherwise normal heart. Three years later EC. showed repetitive flutter.*

History. Sudden attacks of rapid heart-beating for a few seconds, minutes, or hours. More frequent when lying, occasionally a dozen a day. Referred by a doctor because of extrasystoles.

Examination (October 1941). Pulse 120 with occasional irregularities. At times 5–6 beats in paroxysms at a rate of 160–180. Otherwise no cardiac abnormality. No EC. then recorded.

In December 1944 she had the same complaints. EC. basic rate 90–100, regular, with tall P II. Sudden bursts of slightly irregular, apparently independent ventricular activity of 200, lasting for 7–12 auricular cycles, after which the normal rhythm resumes without intervening pause. CR_1: typical flutter waves (Fig. 8).

Case 30, *aged* 22.

Woman of 22 *had irregular heart when aged* 6; *normal adolescence. Palpitation on effort. EC. during two years of observation always showed nodal tachycardia on exertion, nodal rhythm or sinus rhythm at rest. No other signs of heart disease. Spontaneous improvement with little acceleration on exertion, but persistence of abnormal rhythm.*

History. When aged 6, heart was found irregular: she was allowed to play games only after the age of 13. For 2–3 months, palpitation and increasing dyspnœa on exertion.

Examination (July 1944). On standing, pulse 120, regular; after ½–1 minutes irregular, then 76, regular. Exercise increases rate to 130, persisting for several minutes. No other abnormal signs; radioscopy normal. EC. nodal tachycardia.

Course. EC. were recorded first at fortnightly, then at monthly, later at three-monthly intervals from July 1944 till June 1946. At rest: nodal rhythm, no P waves visible, rate 60–70, regular. Sinus rhythm found only once. On exertion: nodal tachycardia, 130–140, regular; when rate drops few sinus beats appear. At the last examination there was sinus rhythm in lead I, nodal rhythm in leads II and III while at rest, nodal rhythm at a rate of 96 on exertion. She was then better and able to exert herself without much palpitation or dyspnœa.

Case 32, *aged* 35.

Woman, aged 35, *complaining of sudden faintness and palpitation. Pulse rapid, irregular; otherwise normal heart. EC. during* 2½ *years of observation constantly showed repetitive ventricular tachycardia and multiform extrasystoles. Quinidine ineffective. Ten years later mild toxic goitre, yet no recurrence of paroxysms.*

History. Well until one year ago when she collapsed in the street with sudden palpitation, weakness, and dizziness; many similar attacks since.

Examination (April 1921). Except for irregular pulse nothing abnormal. No cardiac enlargement on radioscopy. EC., R.V.P.T., rate 220 reg.; aur. rate 150, reg. Shorter runs of 2–3 V.Ex. Intervening sinus beats at a rate of 110 (Fig. 11A and B).

Course. EC. recorded twice in February 1922, June 1922, October 1923, always showed R.V.P.T. at a rate of 200–250. Quinidine at first reduced the number of attacks, but was later ineffective. Seen again January 1931, still complained of palpitation. Examination then showed small toxic goitre, with a pulse rate of 96. B.P. 160/90, slight tremor; normal heart on physical and radiological examination. EC. showed N.R. except for single ventricular extrasystoles. She improved with rest and sedatives and the signs of toxic goitre subsided. When last seen in 1934, she felt well; single V.Ex. were still present in the EC.

Case 33, *aged* 20.

Man of 20, *served as air pilot during the war; discharged from R.A.F. because of heart disease. Complains of palpitations and fainting attacks. Normal heart except for tachycardia. Three EC. during* 10 *months of observation as an out-patient showed regular ventricular tachycardia, rate* 120. *While in hospital and on digitalis, transient sinus rhythm appeared, followed by repetitive ventricular tachycardia, always present during the remaining* 14 *months of observation.*

History. Two years ago when volunteering for the R.A.F. pulse was found "queer." Examined several times before being accepted Grade 1. Trained as a pilot, apt for his duties, flying at times at 20,000 feet without oxygen. Tonsillitis 6 months ago; kept for 3 months in hospital where X-rays and EC. were taken. Discharged from R.A.F. because of "congenital heart disease." Complains of frequent palpitation and dizziness. Fainted four times during the year while on parade.

Examination (November 1943). Pulse 110, reg. B.P. 105/80. Heart sounds normal. Radioscopy: full-sized heart within normal limits. EC., V.T. (Right B.B.Bl.), 125 reg., no P waves seen Five days later, EC. similar.

Course. Seen again September 1944; having frequent palpitation even at night, usually short attacks 1–2 min. duration. EC., V.T. as before. Under observation at the London Hospital 9th October till 11th November 1944. V.T. persisted while at rest, rate 110–130. Under digitalis leaf 1½ grains t.i.d. pulse became irregular and the rate decreased occasionally to 50 when the pulse became regular and the EC. normal. Digitalis, 1 grain bis die, later t.i.d.; successive EC. always showed V.Ex. singly or in short runs (3–5), at a rate of 120–130, preceded by a sinus beat and followed by pause with a basic rate of 90–96 (Fig. 12). One month later the EC. was the same. In 1946 it was heard by chance that he had committed suicide.

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A CASE OF PERFORATED DUODENAL ULCER AND CARDIAC INFARCTION

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The following case was thought to be of sufficient interest to be worth reporting because cardiac infarction seemed to follow within an hour of a perforated duodenal ulcer.

A musician, aged 58 years, was admitted to Edinburgh Royal Infirmary, on 21/1/47. For six months he had experienced attacks of pain over the upper part of the sternum, crushing in character, and radiating to the shoulder and arm as far as the elbow on the left side. The pain was brought on by exertion, over-eating, excitement, and cold; and was relieved by rest, leaving the præcordium and the left arm numb. The attacks became progressively more severe, more frequent, and more easily provoked by exertion.

Three months from the onset he experienced a violent attack of sternal pain while at rest in bed; the pain was very severe, crushing in character, and radiated down the left arm to the fingers. It persisted all night but eased next day. He was not confined to bed at this time but remained in the house resting. During the following months he had several further attacks of pain, usually brought on by very slight exertion, such as washing or dressing. Electrocardiograms in November 1946, showed little abnormality in the resting records apart from left axis deviation, but conspicuous S–T distortion after exercise.

The patient had suffered periodically from indigestion for 20 years. The attacks were characterized by pain and distension in the epigastrium, relieved by food and alkalis. A barium meal examination three years before had shown no abnormality.

The family and social history was irrelevant.

On examination he was of average height and build, anxious and worried, but without cyanosis or œdema. Clinical examination showed slight cardiac enlargement, a blood pressure of 180/110, faint heart sounds with gallop rhythm and a soft localized apical systolic murmur. Radiologically there was slight enlargement of the left ventricle.

Abdominal examination showed no restriction of movement and no palpable mass or tenderness, but revealed guarding of the upper right rectus muscle. No other abnormalities were discovered on physical examination. The blood count was normal, the Wassermann reaction and urine examination negative. An electrocardiogram taken on 22/1/47 showed no gross abnormality.

The clinical diagnosis was angina pectoris with severe coronary arterial disease. The patient was treated with complete rest in bed, a low residue diet, and half a grain of phenobarbitone morning and night, and glyceryl trinitrate tablets, 1/100 of a grain, taken when required.

Progress. One week after admission his condition showed little change: even turning in bed brought on an attack of angina pectoris.

At 12 noon on 29/1/47 he suddenly cried out with pain and lay completely motionless with flexed knees. His pulse was 90 a minute, the temperature 96° F., the respiration rate 28 a minute, and the blood pressure 90/50. Examination revealed entire absence of abdominal

movement on respiration and marked muscular guarding of the upper abdomen with extreme tenderness on palpation in the epigastrium. There was slight diminution of the liver dullness. The heart sounds were faint.

The provisional diagnosis of perforated peptic ulcer was confirmed by a surgeon, but in view of the patient's poor general condition it was decided to adopt conservative rather than operative treatment.

One hour later the patient stated that the pain had spread over the left side of the chest and down the left arm to his fingers. Previously immobile, he was now extremely restless and was bathed in a cold sweat. His pulse had risen to 100 a minute but respiration was unchanged in type and rate. The findings on abdominal examination were similar to those an hour before. Electrocardiograms at 1.20 p.m. and 4.20 p.m. showed changes indicative of recent cardiac infarction (Fig. 1, B and C).

In view of the cardiographic evidence and the history of coronary disease, the provisional diagnosis of perforation was revised. It was assumed the patient had another cardiac infarction. He was treated with large doses of morphia and absolute rest.

During the following six days the patient felt more comfortable, and had little discomfort after the first 48 hours. His pulse, however, continued fast (115–120), his gallop rhythm persisted, and basal crepitations were heard. There was now fever (101° F.) with slight leucocytosis (10,800–11,400) and a fast B.S.R. (24–30 mm. Westergren). Abdominal guarding and epigastric tenderness persisted. The cardiographic changes are discussed later and in more detail in the legend to Fig. 1.

On 6/2/47 the patient began to suffer from cardiac asthma. His pulse rate rose to 130 a minute and he developed coupled beats. The respiratory rate was 36 a minute. The apex beat was 3 cm. outside the mid-clavicular line in the sixth left interspace. The heart sounds were feeble, there were many extrasystoles, and the apical systolic murmur persisted. Dullness on percussion and coarse crepitations were elicited on examination of the bases of the lungs. The patient was treated with continuous oxygen, aminophyllin, and morphia, but death occurred 10/2/47 from left ventricular failure.

Electrocardiographic changes. The routine curve taken at 11.40 a.m. on 29/1/47 is of a type common in cases of myocardial disease of coronary origin, the conspicuous abnormalities being the T inversions in leads I and IVF : it was the same as earlier records.

The record taken at 1.20 p.m. shows marked S–T shifts in chest leads indicative of "injury currents"; the nature of the changes with the history of pain rendered an ischæmic lesion likely. In the record three hours later QRS changes in limbs and chest leads, though slight, are apparent and were read as confirming infarction. The appearance of a small Q in the apical lead, together with the grossly aberrant QRS in leads C_2F suggested an early anterior infarction.

The subsequent records are consistent with a developing infarct and the deep Q in the apical lead is characteristic of such a lesion underlying that electrode. The cardiographic diagnosis was a large coronary infarction involving *inter alia* the apical region.

Necropsy. This was performed on 11/2/47.

The pericardial sac contained 150 ml. of blood-stained serous fluid. The visceral pericardium showed a loss of lustre over the anterior, postero-inferior, and apical regions of the heart. A fibrinous exudate and areas of fibrous thickening were present in the posterior part of the pericardial sac. The heart weighed 400 g.; it was enlarged and showed generalized bulging of the apex and lateral aspect of the left ventricle. The anterior surface, apex, and lower part of the postero-inferior aspect of the heart were the seat of infarction, and internally this extended to the left ventricular wall and the interventricular septum. The myocardium was necrotic, thin, and covered with ante-mortem thrombus. The chambers and auriculoventricular orifices were dilated. The valves were healthy. The coronary arteries were

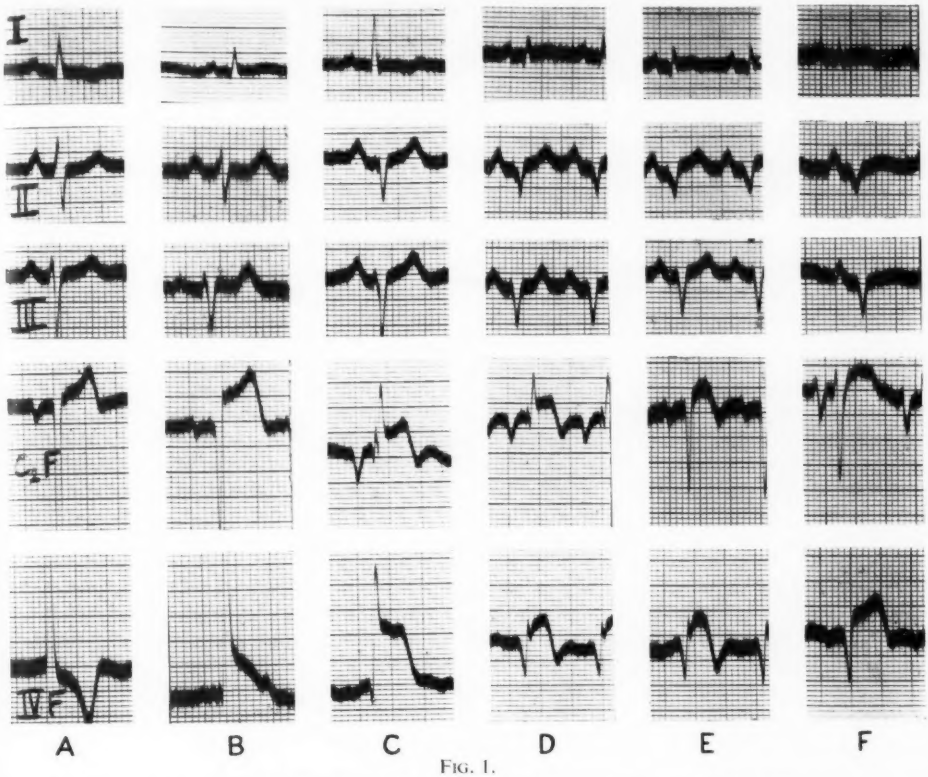

FIG. 1.

(A) 29/1/47, 11.40 a.m. Sinus rhythm, rate 98 a minute; P–R interval 0·18 sec.: left axis deviation; shallow inversion of T in lead I, upright T waves in leads II and III. The sternal chest lead (C_2F), shows a deep Q, and trace of S–T elevation and upright T waves: the T waves in lead IVF are deeply and sharply inverted.
(B) 29/1/47, 1.20 p.m. Sinus rhythm persists with P–R of 0·16 sec., and left axis deviation. The T waves in lead I are now low upright. In lead C_2F S–T elevation exceeding 5 mm. in height has developed without changes in QRS or T. In the apical lead a tiny initial Q precedes the tall R, marked S–T elevation is present, and the inverted T waves have disappeared.
(C) 29/1/47, 4.20 p.m. In this record further changes are apparent, viz.: disappearance of R in lead II; increase in amplitude of R in lead I; diphasic T waves in lead I; gross changes in form of QRS in lead C_2F, with increase in size of vestigial Q in lead IVF; conspicuous "coronary" S–T–T segments in both chest leads.
(D) 30/1/47. Sinus tachycardia rate approx.: 120 a minute: marked decrease in voltage of QRS an all limb leads; persistence of splintered upright QRS in lead C_2F; development of deep Q as main deflection in lead IVF; beginning inversion of terminal position of T in both chest leads.
(E) 31/1/47. The principal changes from the last record are increased slurring of QRS in limb leads; shallow inversion of T in lead I; steep downward initial deflection in lead C_2F; and increase in depth of T inversion in both chest leads.
(F) 10/2/47. This record shows further reduction in voltage in limb leads, involving QRS and T waves; return of initial deflections in lead C_2F to an rS pattern, and conspicuous if in the apical lead, with S–T elevation and upright T waves in both chest leads.

atherosclerotic with greatly narrowed lumina. Recent ante-mortem thrombus was present in the descending branch of the left coronary artery. The aorta showed minimal atherosclerosis in its descending portion only.

An abscess was present in the peritoneal sac, localized in the space between the hepatic

flexure, liver, and duodenum, walled off from the peritoneal cavity by dense fibrous adhesions and containing 200 ml. of thick, foul smelling, greenish yellow pus. There was no generalized infection of the peritoneum. The abscess had originated from the perforation of a chronic peptic ulcer situated in the duodenum. The duodenum presented a peptic ulcer at the antero-inferior aspect of the first part immediately beyond the pylorus. The crater, measuring 12 mm. × 5 mm. had a hard sclerotic base and firm edges. A small perforation had occurred at the upper end, the lower extremity had eroded into the head of the pancreas.

The appearance of the myocardial infarct and the localized peritonitis indicated they were both of about 14 days' duration.

There were no other relevant findings apart from bilateral hydrothorax and congested and œdematous lungs.

Discussion

When the case is reviewed in the light of the post-mortem findings a number of interesting points arise.

The Duodenal Ulcer. Despite the complaint of intermittent indigestion for 20 years, the symptoms were mild, and hardly suggestive of duodenal ulcer, still less of an ulcer eroding into the head of the pancreas; the constant dull boring pain characteristic of this complication of peptic ulcer was absent. X-ray examination three years ago had been negative: the ulcer may have developed since then, though its size and character suggested that it was of long standing.

The Diagnosis and Sequence of Events. The sudden onset of epigastric pain without radiation to the arm, as had occurred on each previous attack of angina; the rigidity and tenderness in the upper abdomen; the diminution of liver dullness; the thoracic type of respiration with grunting expiration; the severe shock; the attitude and immobility of the patient; the relatively slow pulse and the furred tongue, all supported a diagnosis of perforated peptic ulcer, especially with a history of indigestion. The occurrence an hour later of pain in the chest with radiation to the left arm, intense restlessness, rapid pulse, enlargement of the heart, and characteristic changes in the electrocardiogram, all indicated the occurrence of a myocardial infarct.

It is suggested that the perforation occurred at the time the patient suddenly cried out; and, due to the shock, low blood pressure, and poor flow blood, thrombosis occurred in the descending branch of the left coronary artery, that was already the seat of gross atherosclerosis. This was the cause of the extensive myocardial infarction, later causing left ventricular failure and leading to death.

The Perforated Duodenal Ulcer. Post-mortem, the abscess was well localized between liver, hepatic flexure, and duodenum, and was completely walled off from the peritoneal cavity. If the patient had not died from coronary thrombosis the perforated ulcer would presumably have healed by conservative treatment, thus supporting the views of Turner, Taylor, and Visick, all of whom have advocated a conservative régime for these cases.

I wish to thank Professor D. M. Lyon for his permission to publish this case and for his help in the compilation of this paper, Dr. I. G. W. Hill for his help and report on the electrocardiograms, and Dr. L. G. Leitch for the post-mortem findings.

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PAROXYSMAL HEART BLOCK IN BUNDLE BRANCH BLOCK

BY

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When a patient is suspected of having Stokes-Adams disease the electrocardiogram is of critical importance, particularly in respect of the auriculo-ventricular (A-V) conduction time. Should this not be prolonged, the possibility of paroxysmal heart block remains, although the diagnosis of Stokes-Adams disease is uncertain without some degree of heart block recorded graphically. It is not known how far major impairment of intraventricular conduction (bundle branch block) with a normal P–R interval predisposes to A-V dissociation, and, therefore, to Stokes-Adams attacks. This matter is here considered.

The Investigation

In the records of the Cardiac Department of the London Hospital covering the last fifteen years were found 112 patients over the age of 40 years with heart block of a degree exceeding

TABLE I

Cases without Bundle Branch Block

P–R intervals tabulated with the time relation of electrocardiograms in normal rhythm and in A-V block.

Case No.	Age	Electrocardiograms (EC.)			Clinical comment
		Normal Rhythm P–R (sec.)	Time in relation of EC. in N.R. to EC. in A-V block	Degree of A-V block	
1	57	0·20 0·26 0·24 0·22	29 months before. 9 weeks after. 1 year after. 4 years after.	C.H.B.	"Giddy attacks" before the first EC.; later Stokes-Adams attacks. For this analysis 0·26 sec. is taken as the P–R interval.
2	63	0·32	Same day.	C.H.B.	Stokes-Adams attacks.
3	61	0·17	9 months before.	C.H.B.	Epileptiform attacks for 8/12 before EC. with normal rhythm.
4	63	0·27	Same EC.	2 : 1 H.B.	Referred from Neurological Dept. Stokes-Adams attacks.
5	62	0·28	Same EC.	2 : 1 H.B.	Referred from Neurological Dept. Syncope.
6	49	0·30	15 months before.	C.H.B.	Stokes-Adams attacks.
7	60	0·24	7 years before.	C.H.B.	Stokes-Adams attacks.
8	43	0·19	Same day.	C.H.B.	Epileptiform attacks; referred from neurologist. Five years later L.B.B.Bl., C.H.B.
9	41	0·22 QRS normal	6 weeks after.	C.H.B. R.B.B.Bl.	Stokes-Adams attacks continued when 3 years later EC. showed normal QRS and P–R varying from 0·2 to 0·24.
10	55	0·30 0·40	Same day. 10 days before.	C.H.B.	Stokes-Adams attacks.
11	52	0·30	13 days before.	2 : 1 H.B.	Stokes-Adams attacks.
12	46	0·29	1 month before.	2 : 1 H.B.	Myotonia atrophica. No syncopal symptoms.

a prolonged P–R interval alone. Among these were 81 (62 with complete and 19 with partial heart block) from whom a cardiogram in normal sinus rhythm was never obtained and who were, on this account, discarded from the series. Thirty-one cases had cardiograms showing normal sinus rhythm as well as A-V block (partial or complete) at one time or another. Their ages ranged from 41 to 82 years. Those below 40 have been excluded because, in them, transient heart block may result from an acute infection such as rheumatic fever, whereas the condition now considered arises from ischæmic changes which are, in the main, progressive. For purposes of analysis the 31 cases were separated into three groups.

(1) *A-V Block without Bundle Branch Block*

In this category there were 12 cases. Their cardiograms in normal rhythm contained normal QRS complexes, although in the phase of A-V block one man (Case 9) showed right bundle branch block (*R.B.B.Bl.*). Complete heart block (*C.H.B.*) was demonstrated at least once in 8 of this group, and in the remaining 4 there was 2 : 1 heart block. Heart failure or cardiac infarction, either of which might impede conduction, was absent in all of them. In every case, except one with myotonia atrophica (Case 12), Stokes-Adams attacks preceded the cardiographic investigation. Details of this group are given in Table I.

TABLE II

CASES WITH BUNDLE BRANCH BLOCK, UNCOMPLICATED BY CARDIAC INFARCTION OR HEART FAILURE

Case No.	Age	EC. in Normal Rhythm		Time relation of EC. in N.R., to EC. in A-V block	EC. showing A-V block	Clinical comment
		P–R (sec.)	Other features			
13	57	0·2	L.B.B.Bl.	Same EC.	2 : 1 H.B. L.B.B.Bl.	Attacks of unconsciousness for 3 months before these EC.
				17 days after and twice previously.	C.H.B., L.B.B.Bl.	
14	49	0·2	R.B.B.Bl.	8 months before.	C.H.B., L.B.B.Bl.	3½ years' giddiness and faintness. 22 months falling unconscious.
15	63	0·18	R.B.B.Bl. (newer type).	6 days after.	2 : 1 H.B. R.B.B.Bl.	First Stokes-Adams attack 15 months before these EC.; many observed in hospital.
16	55	0·4	R.B.B.Bl. (newer type).	9 weeks before.	2 : 1 H.B. R.B.B.Bl.	Stokes-Adams attacks.
17	72	0·18	R.B.B.Bl. (newer type).	8 days after.	C.H.B., R.B.B.Bl.	Stokes-Adams attacks for 2 years before these EC.
18	60	0·25	L.B.B.Bl.	8 months after.	C.H.B., L.B.B.Bl.	C.H.B., with Stokes-Adams attacks for 4 years after and symptoms 6 years prior to first EC.
19	51	0·19	L.B.B.Bl.	Same day.	2 : 1 H.B. L.B.B.Bl.	Stokes-Adams attacks for 3 weeks before the first EC.
				8 months before.	C.H.B., R.B.B.Bl.	
20	55	0·2	L.B.B.Bl.	3 months after.	C.H.B., L.B.B.Bl.	Several Stokes-Adams attacks in hospital. Still alive 3 years later in C.H.B. L.B.B.Bl.
21	54	0·18	L.B.B.Bl.	2 years before.	2 : 1 H.B. L.B.B.Bl.	Occasional Stokes-Adams attacks over a period of 5 years; P–R still less than 0·2 sec. after last attack.
22	46	0·2	L.B.B.Bl.	4 months before.	2 : 1 H.B. L.B.B.Bl.	"Faints" for 3½ years before EC. obtained to prove Stokes-Adams disease.
23	51	0·18	L.B.B.Bl.	31 months before.	C.H.B., R.B.B.Bl. (newer type).	C.H.B. with Stokes-Adams attacks for 4 years, which include this interval of 31 months.
24	60	0·22	L.B.B.Bl.	Same EC.	3 : 2 H.B. L.B.B.Bl.	B.P., 210/130. "Dizzy spells." No pain or failure.

(2) *A-V Block with Bundle Branch Block*

This group also comprised 12 cases and they showed the cardiogram of bundle branch block. In sinus rhythm 8 had left bundle branch block (*L.B.B.Bl.*) and 4 *R.B.B.Bl.* of the type more recently recognized (Wilson *et al.*, 1934; Evans, 1937). A change of bundle branch pattern from left to right was seen in 2 cases (Cases 19 and 23) and in one (Case 14) from right to left with the record of A-V block. Particulars of this group are given in Table II.

(3) *With Cardiac Infarction or Heart Failure*

This group contains 7 cases that were separated for two reasons.

Cardiac infarction could not be excluded in 4 (Cases 25, 28, 29, and 30). Such an accident, rapidly causing heart block, could happen regardless of the previous state of the genetic system. In this exclusion particular attention was paid to the history of pain because bundle branch block may mask the electrocardiographic signs of cardiac infarction (Wood, Jeffers, and Wolferth, 1935; Wilson, 1936 and 1945). One of these patients with coronary pain (Case 30) was the only case of this group without bundle branch block.

Heart failure was present in 4 patients (Cases 25, 26, 27, and 31). There is evidence that this factor may precipitate heart block (Willius and Anderson, 1934), and accordingly these cases were separated from the first two groups, which, as will be seen, makes no material difference to the conclusions when the inclusive figures are compared.

Before proceeding to the analysis of electrocardiograms it is necessary to consider the normal P–R interval. According to the Criteria Committee of the New York Heart Association (1943) the upper limit of the normal P–R interval in adults with normal heart rates is 0·2 sec. This arbitrary measurement is adopted for the present analysis. One other point

TABLE III

CASES COMPLICATED BY HEART FAILURE OR POSSIBLE CARDIAC INFARCTION

Case No.	Age	EC. in Normal Rhythm		Time relation of EC. in N.R., to EC. in A-V block	EC. showing A-V block	Clinical comment
		P–R (sec.)	Other features			
25	62	0·21	R.B.B.Bl. (newer type).	3 weeks after.	3 : 1 H.B. R.B.B.Bl.	First Stokes-Adams attack 6 weeks before pain in chest after syncope on day of admission. Several Stokes-Adams attacks observed in hospital. ? Cardiac infarction. Heart failure. Died 1 yr. later in C.H.B.
26	59	0·23	L.B.B.Bl.	6 weeks before.	C.H.B., L.B.B.Bl.	Heart failure from mitral stenosis and aortic incompetence. Repeated periods of C.H.B. under observation.
27	57	0·2	R.B.B.Bl.	1 week after.	C.H.B., R.B.B.Bl.	Hypertensive heart failure. No complaint of syncope.
28	64	0·17	L.B.B.Bl.	5 days after.	2 : 1 H.B. L.B.B.Bl.	Lt. mammary pain for 2 weeks. Frequent "faintness." Not unconscious. P. rate noticed to drop by half on several occasions. Cardiac infarction is not excluded.
29	56	0·19	L.B.B.Bl.	7 weeks after.	C.H.B., R.B.B.Bl.	For 3 weeks before C.H.B. found in EC., præcordial pains at rest and with exertion lasting up to 20 minutes. Cardiac infarction.
30	58	0·2	No B.B.Bl.	16 days before and 9 days after.	C.H.B.	Referred from Venereal Clinic. Angina pectoris. Treated syphilis. Cardiac infarction not excluded, although unlikely.
31	82	0·18	R.B.B.Bl. (newer type).	15 weeks after.	C.H.B., R.B.B.Bl.	Hypertensive heart failure in C.H.B. Regained N.R. after treatment by rest and digitalis.

to be stressed concerning A-V conduction time is that the longest interval in the three limb leads should be taken as the true reading for that cardiogram. This seemed to be found most commonly in lead II, although other observers have considered lead III to be the most reliable single lead for the measurement of the time intervals (White, Leach, and Foote, 1941). In no cases studied in these three groups was there any evidence of ventricular tachycardia or similar arrhythmia, and it is probable that, as usual in paroxysmal heart block, the Stokes-Adams attacks could be classified in Group I of Parkinson, Papp, and Evans (1941) namely, ventricular standstill alone.

Selected Case Histories

Case 13. Female, aged 57, with hypertension. She had fallen unconscious on two occasions in the three months before she was seen in *C.H.B.* Similar attacks recurred while she was under observation during the next four months. In one cardiogram, normal rhythm with a P–R interval of 0·2 sec. was seen in leads II and III although there was 2 : 1 A-V block in lead I (Fig. 1). On other occasions *C.H.B.* was recorded and there was *L.B.B.Bl.* in all tracings.

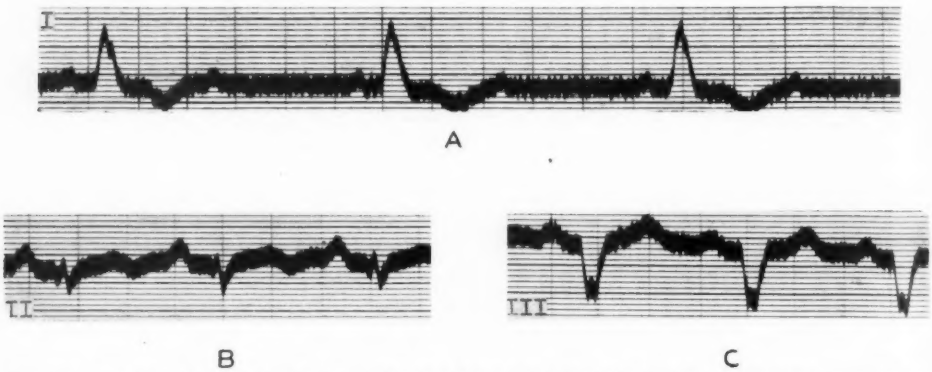

Fig. 1.—*Case* 13. (A) Left bundle branch block with 2 : 1 A-V block in lead I. (B) and (C) normal sinus rhythm in leads II and III on the same occasion.

Case 14. Male, aged 49. Referred from the Neurological Department; he had previously been suspected of having epilepsy. For 3½ years he had been troubled by very short attacks of faintness, and for 22 months he had been falling unconscious, sometimes hurting himself. An aura consisted of giddiness, but there was never tongue-biting or incontinence. No attacks were witnessed in hospital. His cardiogram showed *R.B.B.Bl.* and normal sinus rhythm with P–R 0·2 sec. Because of this normal P–R interval, Stokes-Adams disease was considered unlikely to be the cause of these faints which continued unchanged. Eight months later a cardiogram was obtained which showed *C.H.B.*, and *L.B.B.Bl.* (*N.B.*—Change of bundle branch block from left to right.)

Case 17. Male, aged 72. He reported three isolated attacks of unconsciousness, 24, 18, and 4 months previously, but had suffered repeated faints during the two weeks prior to his admission to hospital. His first cardiogram portrayed *C.H.B.*, and *R.B.B.Bl.* of the common type. Eight days later in normal rhythm his tracing contained a P–R interval of 0·18 sec. In the following month two further cardiograms were obtained with normal sinus rhythm and P–R intervals of 0·2 and 0·18 sec. respectively.

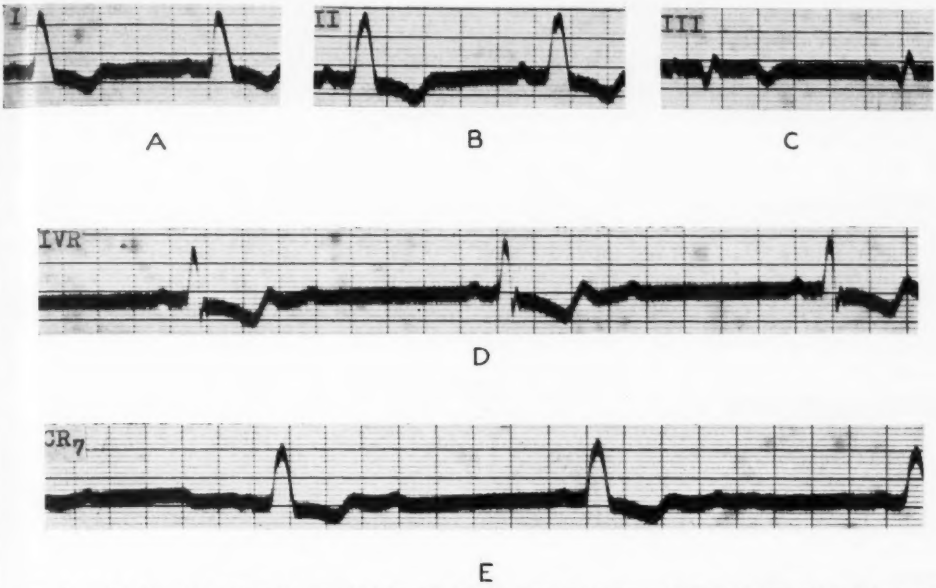

FIG. 2.—*Case* 19. (A), (B), and (C). Left bundle branch block with normal sinus rhythm and P–R interval 0·19 sec., in limb leads. (D) and (E). A few minutes later 2 : 1 A-V dissociation in IVR and CR_7.

Case 19. Male, aged 51. He first complained of dyspnœa on exertion and fainting attacks 12 years before he attended the Cardiac Department. No syncope had occurred in the last 9 years until, in the last three weeks, these bouts had returned with sudden violence. His pulse was regular at 44 to the minute and the blood pressure was 140/80. The limb leads of the cardiogram showed normal sinus rhythm at 60 a minute, *L.B.B.Bl.*, and a P–R period of 0·19 sec. (Fig. 2). When his chest leads were taken a few minutes later, however, there was 2 : 1 A-V block with a ventricular rate of 37. Eight months later there were *C.H.B.* and *R.B.B.Bl.* (*N.B.*—Change of branch block.)

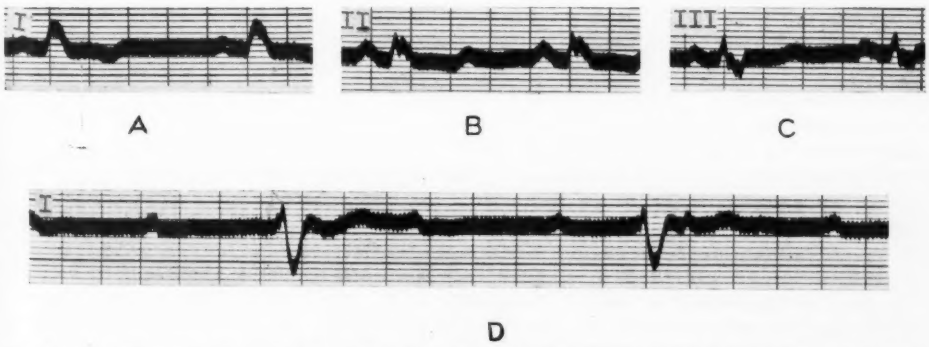

FIG. 3.—*Case* 23. (A), (B), and (C). Left bundle branch block, normal sinus rhythm, and P–R interval 0·18 sec. (D) Thirty-one months after. Complete heart block and right bundle branch block.

Case 31. Female, aged 82. This patient had *C.H.B.* and *R.B.B.Bl.* when she was first examined. The diagnosis of hypertensive heart failure was confirmed radiologically. After treatment by rest and digitalis, she regained normal rhythm. Fifteen weeks after the first cardiogram, the P–R period was 0·18 sec. with *R.B.B.Bl.*, and the hilar regions were clear of congestion on cardioscopy.

Results

Among the 13 cases with normal QRS complexes there were 3 (Cases 3, 8, and 30) whose electrocardiograms in normal sinus rhythm showed P–R periods measuring 0·2 sec. or less. In the combined groups there were 18 cases with bundle branch block. Thirteen of these, when in normal sinus rhythm, showed normal P–R periods. If one disallowed those cases that were complicated by heart failure or by possible infarction, the percentage with normal

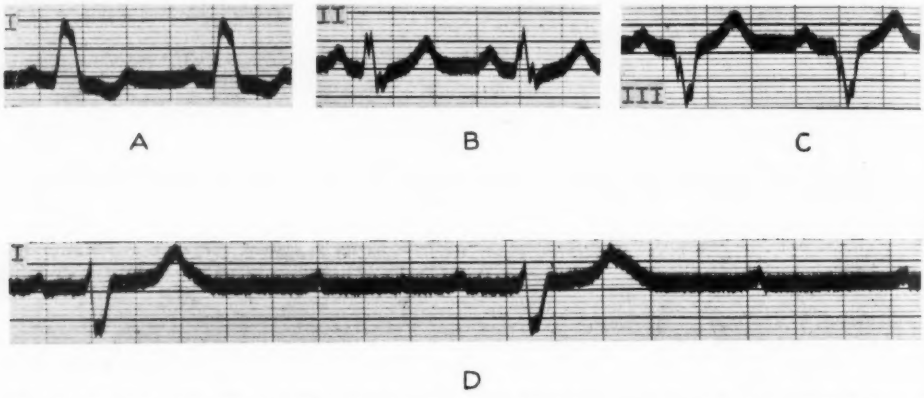

Fig. 4.—*Case* 29. (A), (B), and (C). Left bundle branch block in normal sinus rhythm with P–R interval 0·19 sec., seven weeks after complete heart block with right bundle branch block (D).

A-V conduction time was the same. Adding those with heart failure, but still excluding the patients who may have suffered infarction, one found that this figure was about the same. It would therefore appear that the presence of bundle branch block increases the tendency to A-V dissociation in patients with normal P–R intervals, and that in cases of paroxysmal heart block, a prolonged P–R interval is less often recorded in the phase of normal sinus rhythm when bundle branch block is present than when it is absent from the cardiogram.

Discussion

In clinical medicine there has been a tendency to consider the bundle branches as separate from the main auriculo-ventricular stem, in spite of experimental facts that suggest that the conduction system should be studied as a whole. First, the transmission of impulses in the bundle branches is included in the A-V conduction time. Secondly, the effects of bilateral bundle branch lesions require consideration. Wilson and Hermann (1921) remarked that lesions of the branches of the bundles of His had so far been shown to produce but two kinds of disturbances, namely complete bundle branch block and incomplete bundle branch block, the latter corresponding to delayed conduction in the main stem. They concluded from experiments that when one branch of the bundle is interrupted, lesions of the remaining branch have the same effect as lesions in the main stem. Scherf and Shookhoff (1925) by cutting one bundle branch and compressing the other between its upper and middle third, were able to

produce various degrees of A-V block, ranging from simple prolongation of the P–R period to marked second degree or transient complete block. Our knowledge today is well summarized by Dressler (1945). A disturbance of conduction that involves equally both bundle branches, produces electrocardiographic changes indistinguishable from those caused by lesions of the main stem of the bundle. Interruption of conduction in both bundle branches brings on complete A-V dissociation; or, if there is an equal delay, as opposed to complete block, in both bundles, it is added to the A-V conduction time causing prolongation of the P–R period. As connecting pathways between the bundle branches and between the common trunk and the interventricular myocardium are found in many hearts, the A-V conduction time occasionally may be normal in spite of destruction of both branches. This has been termed " bilateral missed block " (Maheim, Winston, and Roesler, 1943). From histological examinations of the conducting system in cases of intraventricular block, it is known that both bundle branches are usually involved (Maheim, 1931; Yater, Cornell, and Clayton, 1936; Yater, 1938). It is also understood that depression of conduction in either one or both bundle branches may occur with little structural disease (Sigler, 1944). Variations of conduction in the bundle branches are now recognized more frequently (Comeau, Hamilton, and White, 1938). Variable ventricular complexes in heart block and their relation to bilateral bundle branch block were discussed by Bain (1944). Mathewson (1913) who first described such complexes, thought that in his case the impulse was conducted alternately by each branch. Cases of sinus rhythm with intraventricular block in which the type of ventricular complex varied, have been reported by many authors, reference to whom was made by Strauss and Langendorf (1943). More recently alternating bundle branch block has been discussed by Alvarez *et al.* (1944), and Dressler (1945) described two cases in which the P–R interval increased with changes from left to right intraventricular complexes. This variation in conductivity of the bundles, together with the facts mentioned, strongly support the belief that a sudden increase of impairment in both bundles, amounting to bilateral block, may cause auriculo-ventricular block.

In the experience of Campbell (1943), the association of bundle branch block with latent heart block (prolonged P–R interval) was much more common than would be expected if it were a chance coincidence. In 1944 he commented on the closer association between bundle branch block and complete heart block, intraventricular lesions being present in nearly 30 per cent of his series of complete heart block. Although by themselves such associations prove nothing, they are to be expected if there is any truth in the theory of occasional A-V dissociation through bilateral bundle branch block.

Conclusion

The electrocardiograms of 31 patients over 40 years of age, with sometime A-V dissociation (2 to 1, or complete) were specially examined for the presence of bundle branch block, and for the length of the P–R period at a time when there was sinus rhythm. Bundle branch block was present in 18 and absent in the other 13 patients. In the 18 cases with bundle branch block, the P–R period was normal in 13, and long in 5. In the 13 cases without bundle branch block the P–R period was normal in 3, and long in 10.

Thus, paroxysmal heart block is more likely to occur in patients with a normal P–R period if there is bundle branch block than if the QRS complexes are physiological. This observation is of some clinical importance in the diagnosis of Stokes-Adams disease when the cardiogram does not show A-V block.

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THE FIRST HEART SOUND IN AURICULAR AND VENTRICULAR EXTRASYSTOLES

BY

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It is well known that the first heart sound of a premature beat may be of greater or lesser intensity than the first sound of the preceding or following normal beats (Cossio, 1944). An increased intensity of the extrasystolic first sound might be due to the onset of a premature ventricular beat coinciding with the auricular systole of sinus origin (Lewis, 1935), but this explanation could only apply to an extrasystole of ventricular type. As Cossio and others (1943, 1944, 1945) have shown that an increased intensity of the first sound depends on the position and tension of the auriculo-ventricular valves at the onset of ventricular systole, it was decided to investigate the behaviour of the first sound in premature beats and to determine its clinical significance.

Present Investigation

Simultaneous electrocardiographic and phonocardiographic records, with a camera speed of 75 mm. a second, were obtained from 30 patients with extrasystoles. First, the auricular or ventricular origin of the premature beats, and their situation in diastole, that is their degree of prematurity, were established; and also the time relation of the ventricular systole of the premature beat to the premature auricular systole in the case of auricular extrasystoles, and to the auricular systole of sinus origin in the case of ventricular extrasystoles. Secondly, the intensity of the first sound of the premature beat was determined by phonocardiogram and compared with that of the preceding and following normal beats. The time interval between the onset of ventricular systole and the first sound (onset of QRS to first sound) was determined both in the premature and the normal beats.

Of the 30 cases investigated, 16 proved to have auricular and 14 ventricular extrasystoles.

Auricular Extrasystoles

In all but one of the 16 cases (Table I), the extrasystolic first sound was always louder than the first sound of the preceding and following normal beats. The time interval between the onset of QRS and the reinforced extrasystolic first sound varied from 0·05 to 0·08 seconds, while in the case of normal beats or premature beats without reinforcement of the first sound, it was 0·03 to 0·05 seconds. Thus in relation to QRS, the reinforced extrasystolic first sound occurred from 0·01 to 0·05 seconds, average 0·03 seconds, later than the first sound of normal beats.

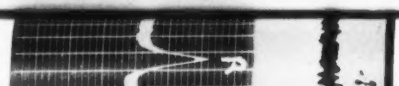
The increased intensity of the extrasystolic first sound and its corresponding delay bear a close relation to its degree of prematurity. The greatest intensity and delay of the first sound was observed when the extrasystolic ventricular systole occurred some time after the T wave of the preceding cycle, that is in mid-diastole (Fig. 1). In contrast, the first sound was less intense, even less than that of normal beats, when the extrasystolic ventricular systole occurred

TABLE I

PHONOCARDIOGRAPHIC AND ELECTROCARDIOGRAPHIC FINDINGS IN 16 CASES OF AURICULAR EXTRASYSTOLES

Case	Normal beat		Premature beat		First sound normal beat		First sound premature beat		Remarks
	R–R interval in seconds	P–R in seconds	R–R interval in seconds	P–R in seconds	Intensity	Onset of QRS to 1st sound	Intensity	On set of QRS to 1st sound	
1	0·69	0·18	0·57	0·20	+	0·03	+++	0·08	
			0·44	0·20			+++	0·07	*
2	0·74	0·20	0·52	0·20	+	0·04	++	0·05	*
3	0·68	0·14	0·44	0·14	++	0·03	+++	0·07	*
4	0·72	0·18	0·44	0·20	++	0·04	+++	0·06	*
			0·52	0·15			++	0·05	*
5	0·68	0·16	0·46	0·14	+	0·04	+++	0·07	*
6	0·88	0·12	0·64	0·14	+++	0·05	++	0·05	
			0·38	T+P			+++	0·07	*
7	0·48	0·16	0·42	0·16	+	0·05	++	0·06	
8	0·48	0·16	0·41	T+P	+	0·04	++	0·06	
			0·48	0·08			+++		Split extrasystolic first sound.
9	0·70	0·14	0·52	0·08	++	0·05	+++	0·06	
			0·60	0·20			++		(*x*) Accurate measurement not possible.
10	1·08	0·20	0·48	T+P	+	(*x*)	++	(*x*)	
			0·51	0·18			+++	0·06	
11	0·88	0·16	0·40	P+T	++	0·04	+++	0·08	Wandering pace-maker.
			0·48	T+P			+++	0·06	*
12	0·66	0·16	0·52	0·18	++	0·03	+++	0·05	
13	0·56	0·15	0·41	0·21	++	0·04	+++	0·07	*
14	0·75	0·14	0·56	0·16	++	0·04	+++	0·06	
15	0·92	0·14	0·56	0·16	++	0·04	+++	0·07	*
16	0·68	0·15	0·52	0·12	++	0·03	+++	0·06	

* Premature beat's first sound coincides with the rapid inflow phase.



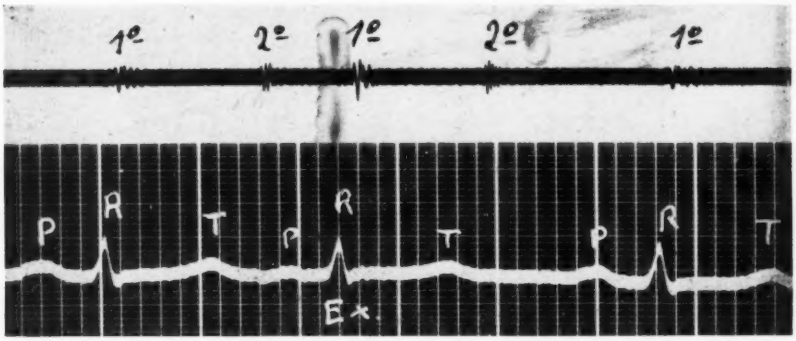


FIG. 1.—Simultaneous phonocardiogram and electrocardiogram showing auricular premature beat falling in mid-diastole; the first sound is intensified and delayed.

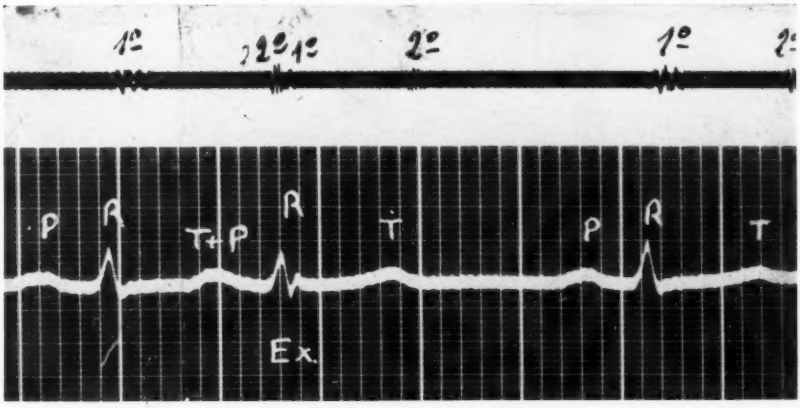


FIG. 2.—Auricular premature beat occurring early in diastole, with a fainter first sound than that of normal beats.

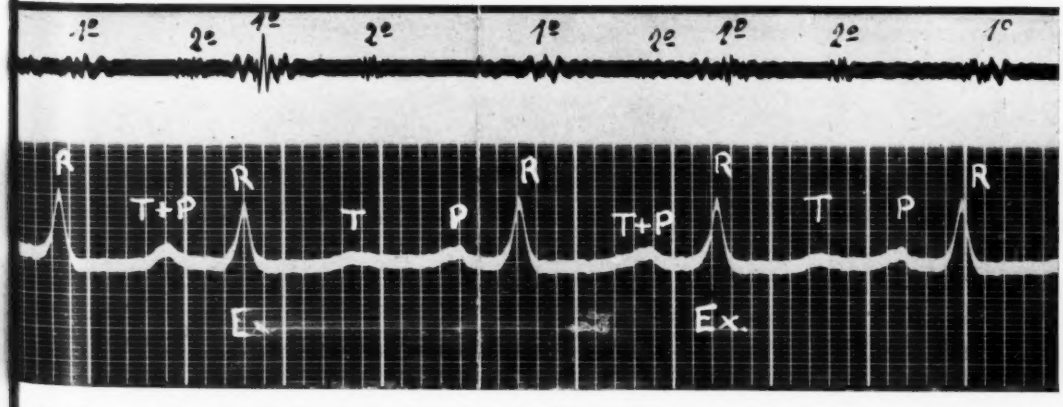


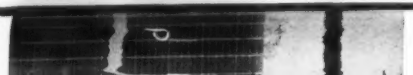
FIG. 3.—Two auricular premature beats are shown: the first occurring in mid-diastole has an intensified and delayed first sound; the second falling later in diastole has a first sound of normal intensity and normal time relation to QRS.

TABLE II

PHONOCARDIOGRAPHIC AND ELECTROCARDIOGRAPHIC FINDINGS IN 14 CASES OF VENTRICULAR EXTRASYSTOLES

Case	Normal beat		Premature beat		First sound normal beat		First sound premature beat		Remarks
	R–R interval in secs.	P–R in in secs.	R–R interval in secs.	P–R in secs.	Intensity	Onset of QRS to 1st sound	Intensity	Onset of QRS to 1st sound	
1	0·72	0·20	0·64 0·58	0·12 0·06	+	0·06	++ +++	0·08 0·12	
2	1·02	0·18	0·56 0·51		+++ ++	0·06 0·05	++ ++ (redpl.)	0·12 0·08	P at end of extrasystolic QRS. P immediately after extrasystolic QRS. Interpolated extrasystolic.
3	0·88	0·12	0·40		++	0·05	++ (redpl.)	0·11	
4	0·70	0·14	0·52		++	0·05	+++	0·11	P in ascending branch of extrasystolic QRS.
5	0·88	0·16	0·44		++ (redpl.)	0·04	++ (redpl.)	0·09	P immediately after extrasystolic QRS.
6	0·72	0·36	0·48		++	0·06	+++	0·12 0·10	P immediately before extrasystolic QRS. (1) Prolonged sound with preponderance of low-pitched components. (2) Prolonged sound with both low-pitched and high-pitched components in equilibrium.
				0·08			+++	0·05	
			0·38				+++ (1)	0·10	
7	0·56	0·22	0·36 0·36		++	0·04	+++ (2)	0·05 0·12	
8	1·02	0·20	0·56		++	0·06	+++	0·10	P immediately after extrasystolic QRS.
			0·56				++	0·10	P immediately after extrasystolic QRS. Interpolated prolonged sound (1).
9	1·20	0·16	0·60		+	0·05	++ (1)	0·10	
10	0·88	0·18	0·64		++	0·06	+++	0·10	P in ascending branch of extrasystolic QRS.
11	0·80	0·22	0·52		+	0·06	++	0·12	P immediately after extrasystolic QRS.
12	0·86	0·16	0·64		+	0·05	++	0·06	P shortly after extrasystolic QRS.
13	0·60	0·16	0·44		+	0·44	++	0·08	P in ascending branch of extrasystolic QRS.
14	0·56	0·14	0·48 0·40		++	0·03	+++ + (redpl.)	0·07 ?	P in ascending branch of extrasystolic QRS. P shortly after extrasystolic QRS. Reduplicated extrasystolic first sound.

redpl. = reduplicated sound.



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immediately after the T wave, that is in early diastole, or when it occurred very late in diastole (Fig. 2 and 3). The A-V conduction time in premature beats with an increased first sound appeared to be longer than that of normal beats, but it could not always be determined exactly, as the extrasystolic P wave was sometimes fused with the preceding T wave.

Ventricular Extrasystoles

In 14 cases investigated (Table II and Fig. 4–7), the extrasystolic first sound was louder than the normal first sound in 9, of equal intensity in 1, of equal or less intensity in 1, and of less intensity in 3 cases. The increased extrasystolic first sound was always delayed in comparison with the normal first sound. The time interval between the onset of QRS and the increased extrasystolic first sound was from 0·08 to 0·12 seconds, average 0·10 seconds, while in the case of normal beats it was 0·03 to 0·06 seconds, average 0·05 seconds. This delay

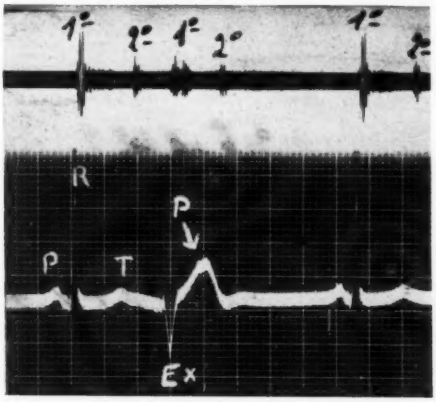

Fig. 4.—Ventricular premature beat falling early in diastole, with faint and reduplicated first sound.

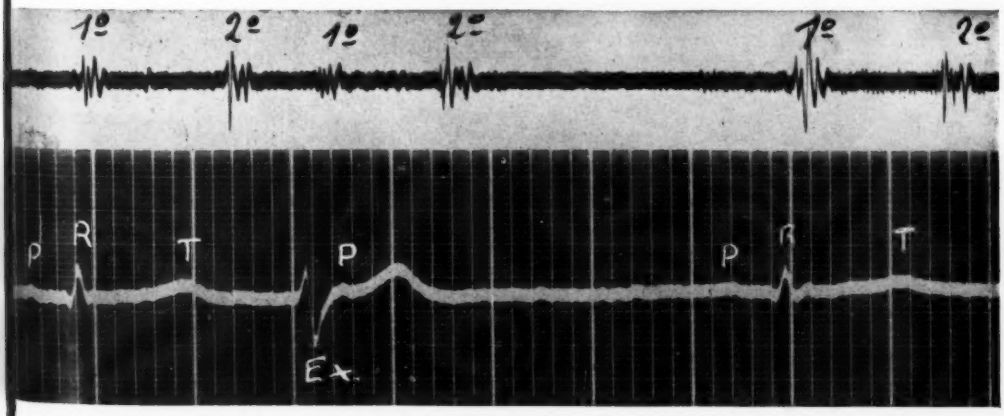

Fig. 5.—Ventricular premature beat falling late in diastole, just before auricular systole, with a faint first sound.

redpl. = reduplicated sound.

is especially evident in the case of the group of rapid vibrations of valvular origin belonging to the first sound, but not in the slower inaudible initial vibrations of muscular origin, which when magnified several times, were always found in the same position.

The increased intensity and delay of the first sound in ventricular extrasystoles was related to their degree of prematurity and to their time relation with the normal auricular systole not followed by a ventricular response. Whenever the premature ventricular systole occurred so early that it coincided with the descending limb of the T wave of the preceding cycle (Fig. 4), or fell later just in front of the next P wave of sinus origin (Fig. 5), the extrasystolic first sound was less intense than that of normal beats. But whenever the premature ventricular systole fell just after the T wave, in mid-diastole (Fig. 6). or just after the next normal P wave (Fig. 7), the extrasystolic first sound was louder than that of normal beats. In 4 of the 14 cases, a split first sound was recorded in the premature beats, though not present in the normal beats.

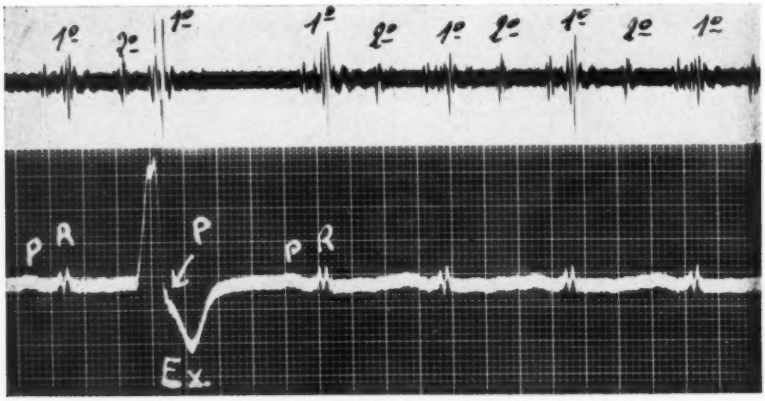

FIG. 6.—Ventricular premature beat, falling, in mid-diastole, with an intensified first sound. Alternation of first sound of succeeding normal beats.

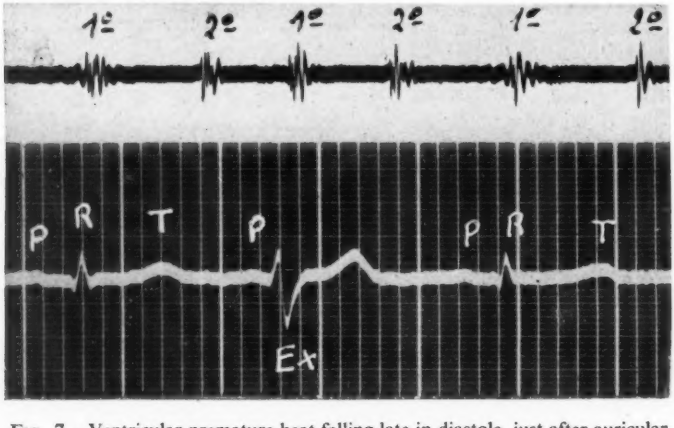

FIG. 7.—Ventricular premature beat falling late in diastole, just after auricular systole, with a slightly intensified and delayed first sound.

Discussion

We conclude from our investigations that intensification of the first sound occurs as often in auricular as in ventricular extrasystoles, and, therefore, cannot serve to differentiate between them. A split first sound, on the other hand, is only produced in ventricular extrasystoles, though not in every case, and its presence suggests that an extrasystole is of ventricular origin. Graphic records show that an intensified extrasystolic first sound is always appreciably delayed in relation to the onset of ventricular systole as recorded in the electrocardiogram. This relationship of the intensity of the first sound and the time of its occurrence has been called the *law of the first sound* (Cossio *et al.*, 1943, 1944, 1945) and it has been attributed to the position and tension of the auriculo-ventricular valves at the onset of systole.

During the cardiac cycle, the A-V valves perform a series of ascending and descending movements with corresponding increase and decrease in their tension. Dean (1936) has shown experimentally that, when blood flows from auricles to ventricles during the phase of rapid inflow, the A-V valves are forced downwards until the maximum opening is attained. As the ventricles fill progressively during diastasis, the A-V valves gradually ascend until the occurrence of auricular systole, when they are again forced downwards, ascending again to a position of almost complete closure on the termination of auricular systole. Normally, the ventricular contraction finds the valves in this position, and their maximum stretch follows immediately; the vibration so produced, which Dock (1933) has shown to be the fundamental cause of the first sound, practically coincides with the onset of ventricular systole.

But, if a ventricular contraction happens to occur at some other moment, for example, at the end of the phase of rapid inflow or during auricular systole, it will find the A-V valves in a much lower position, so that the distance travelled to reach the position of maximum stretch is longer, and consequently more time elapses between the onset of ventricular systole and the valvular vibration that gives rise to the first sound. When the valves have further to travel between the onset of ventricular contraction and the position of maximum stretch, their movement is accelerated, and consequently their vibration is increased, for the kinetic energy is the product of half the mass and the square of the velocity, and the velocity is the product of acceleration and time. This is precisely what has been shown to occur in premature beats, both auricular and ventricular, though more frequently in the former than the latter. When the onset of ventricular systole in a premature beat coincides with an auricular systole of sinus origin, the first sound is intensified and delayed. Similarly, when the ventricular systole of either an auricular or ventricular premature beat falls at the end of or immediately after the phase of rapid inflow, the first sound is intensified and delayed.

Thus there are two propitious moments for the production of the intensified and delayed first sound, at both of which the A-V valves are in their lowest position. When a premature beat falls before the end of rapid inflow, the A-V valves are also lowered, but in this case, owing to incomplete ventricular filling, the valves are insufficiently stretched to intensify the first sound.

There are several reasons why the first sound is more often intensified and delayed in auricular than in ventricular extrasystoles. The first sound represents the summation of vibrations produced by the mitral and tricuspid valves. In auricular extrasystoles this normal summation is unaffected, but in ventricular extrasystoles a synchronism between the two ventricles prevents summation, and the first sound is split. Another reason is that auricular extrasystoles generally fall at a moment propitious for intensification of the first sound. Lastly, ventricular filling is more complete in auricular than in ventricular extrasystoles, because the former have the benefit of an auricular contraction which also contributes to lowering the A-V valves.

Summary

The characteristics of the first heart sound in auricular and ventricular premature beats

have been investigated by phonocardiographic records from 30 cases. In both varieties of extrasystole, the first sound was usually increased in intensity and delayed in relation to the onset of ventricular systole. In ventricular premature beats the first sound was sometimes split. The explanation of these findings is discussed in relation to the position and movements of the auriculo-ventricular valves.

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THE PROGNOSIS OF PATENT DUCTUS ARTERIOSUS

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Surgical intervention is becoming more frequent in patients with a patent ductus arteriosus. At first only marked symptoms were accepted indications for surgery, but these are now becoming much more numerous. This change has largely been due to the gradual decrease of the operative mortality and the dramatic cures effected in cases of bacterial endocarditis. Gilchrist (1946) recommended operation on nearly all uncomplicated cases of patent ductus arteriosus between the ages of 7 and 10 years.

The increased risk that the presence of a patent ductus involves is difficult to assess. Surgeons see the more serious cases with symptoms or with infective endocarditis, and the prognosis without operation in such a series is gloomy. Few physicians have published accounts of large numbers. Maud Abbott's (1936) series based on post-mortem records paints too black a picture as patients with infective endocarditis and congestive heart failure tend to end their lives in hospital. Shapiro and Keys (1941, 1943), Shapiro (1944), Wilson and Lubschez (1942), East (1945), and Gilchrist (1945) have all described series of cases but they do not mention how many were referred for symptoms and how many were symptomless. This is important, as if a large number were referred because of symptoms there must be an excess of the more serious cases which naturally gravitate to hospital or the physician's consulting room.

In the present series the diagnosis of uncomplicated patent ductus arteriosus was made only in the presence of a typical machinery murmur in the second left intercostal space with no evidence of any other abnormality on physical, cardiographic, and *x*-ray examination. Forty-six cases have been collected for analysis and divided into two groups. Group A numbers 30 and, save for one woman, aged 23, referred from an ante-natal clinic because of the presence of a machinery murmur, has been collected from a school cardiac clinic. This must include almost every case of patent ductus arteriosus among the younger people of Bristol, whether they had symptoms or not. Group B consists of 16 cases from areas outside Bristol referred in a few cases from school clinics but often because of symptoms. It follows that Group A forms a representative sample of the condition while Group B approximates more nearly to the type of case generally reported.

Sex Incidence. Of the 60 cases of typical patent ductus arteriosus collected by Shapiro and Keys (1943) 14 were males and 46 females. Steinberg, Grishman, and Sussman (1943) found that of 27 cases 11 were male and 16 female. East (1945) had 4 males and 9 females, Gilchrist (1945) 13 males and 15 females, and Hunter (1945) found 4 males and 10 females. The ratio of males to females varies considerably but in all series there is a preponderance of females varying from slight to very marked. In the present series 13 are males and 33 females. These figures agree substantially with those of other authors.

SYMPTOMS

Maud Abbott (1929) states that a fair proportion are dyspnœic, and that cyanosis, while usually absent, may be present on exertion. Steinberg *et al.* (1943) found 12 of 27 cases

complained of breathlessness. Gross (1939) found his were usually undeveloped and often breathless, but that only those with large ducts had symptoms. Gilchrist (1945) found his patients were often breathless without being aware of it until the patent ductus had been closed, and that in only 5 of 28 was there no limitation of physical capacity. Bullock, Jones, and Dolley (1939) found that only 2 of their 11 cases had no symptoms; all the others had palpitation and were easily tired.

In the present series only 8 children have symptoms at present. Four are slightly breathless on exertion; 3 are easily tired; 5 become slightly blue on occasions; 2 suffer from frequent colds, and only 1 did not play games. In addition 2 children are subject to asthma. Three have more than one symptom. The 30 in Group A include 6 with symptoms, while the 12 surviving members of Group B contain 2 with symptoms. Five of Group B have had bacterial endocarditis and only 1 of these is alive now; this last child was cured by penicillin and is now symptom free. Thus, 7 of the 16 cases in Group B have at some time had symptoms attributable to a patent ductus arteriosus.

Physical Signs

Cases have been described, without murmurs or with systolic murmurs only, that have been proved to have a patent ductus arteriosus post-mortem. Some of these descriptions are of doubtful accuracy as they were before the existence of the typical machinery murmur was recognized, but others are well authenticated. The question of absent murmurs is dealt with later. Descriptions of cases with systolic murmurs only are particularly common before 1910. Typical examples are by Gerhardt (1867), Wasastjerna (1874), Darier (1885), Thomson and Drummond (1900), Simmons (1906), Carpenter (1908), Goodman (1910), Motzfeldt (1913), Wessler and Bass (1913), Weiss (1931), Touroff and Vessell (1940), and Chapman and Robbins (1944).

Shapiro (1944) drew up the following list of the chief findings in cases of patent ductus arteriosus—the presence of a machinery murmur, a systolic thrill in the pulmonary area, an enlarged pulmonary artery, enlarged pulsating pulmonary vessels, a large heart, an increase of the pulse pressure, stunting of growth, absence of cyanosis and of clubbing of the fingers, a normal electrocardiogram, and the presence of heart disease from early childhood. He states that a thrill is usually present (53 of his 62 cases), but may be absent if the ductus arteriosus is small. Gross (1940) also says that a thrill is usually present but that the physical signs of patent ductus arteriosus are frequently not present until the age of 3 years.

In the present series the above criteria have been used in diagnosis. A machinery murmur has been present in all, and all are over 3 years of age. Of the 46 cases 30 had a systolic thrill; 19 of the 30 in Group A had a thrill at some time, but in 3 it vanished with increasing age; in Group B, 11 had a thrill and in 1 it disappeared later. In no case did a thrill become evident if it had been absent earlier. The thrill may have disappeared because the chest wall grew thicker with age, but this does not seem likely as in 2 of the 4 cases examination at intervals of one year showed it to be present on the first and absent at the second occasion. These two lost their thrill by 5 and 6 years of age respectively. In a third case the interval was 4 years. It would seem more likely that some change took place in the volume or mechanics of the circulation.

Blood Pressure

Bullock *et al.* (1939) found that all but one of their 11 cases of uncomplicated patent ductus arteriosus had a high pulse pressure and a low diastolic pressure. Gross (1943) said the diastolic pressure was a measure of the leak and pointed out that among 48 cases, who survived operation for closure of a patent ductus, the diastolic blood pressure which had been low rose to normal after operation. Of their 22 cases, Shapiro and Keys (1941) found only 5 with a pulse pressure of less than 45 and the average was 59. Among normal people they

did not find any pulse pressure higher than 45. Shapiro (1944) said the pulse pressure may be normal if the ductus arteriosus is small. In his 60 cases (including the 22 cases just mentioned) he found the pulse pressure normal in 14, but it was increased in the remaining 46. Tubbs (1945) found the average diastolic pressure was 50–60 mm. Gilchrist (1945) in his series of 28 cases found the average diastolic pressure below the age of 10 years was 48, and the average systolic pressure 95; over the age of 11 years the average diastolic pressure was 53 mm. Judson and Nicholson (1914) found that the average diastolic pressure in normal children of 3 to 15 years ranged between 64 and 71 and the systolic pressure between 91 and 106, the pulse pressure being about 30 mm.

Bohn (1938) measured the diastolic blood pressure of cases with a patent ductus arteriosus after exercise and found it fell to nearly zero. This fall has been substantiated by Shapiro and Keys (1941).

Of the 30 cases for whom blood pressure readings are available only 5 have a pulse pressure of less than 45 mm.; this agrees with the findings of Shapiro and Keys (1941) quoted previously.

TABLE I

THE BLOOD PRESSURE IN PATIENTS WITH A PATENT DUCTUS ARTERIOSUS AND THE EFFECT OF EXERCISE

Age		Number of cases	Average pulse pressure	Average diastolic pressure	Number with a diastolic pressure of 60 mm. or below	Number of cases with a change of diastolic pressure after exercise		
						Fall	Rise	No change
Group A	15 years or below ..	10	53	66	3	3	0	3
	16 years or above ..	11	55	74	1	3	2	4
	Total	21	—	—	—	6	2	7
Group B	15 years or below ..	6	69	68	1	3	1	0
	16 years or above ..	3	58	72	0	0	0	1
	Total	9	—	—	—	3	1	1
Grand total		30	—	—	5	9	3	8

Five cases had diastolic blood pressures of 60 or below; this is contrary to the findings of Tubbs (1945) and Gilchrist (1945) and most of the blood pressure readings come into the range of normal as defined by Judson and Nicholson (1914).

The effect of exercise on the blood pressure is not so easy to obtain as has been suggested. Children and even adults often refuse to exercise to the point of distress. In 3 the diastolic blood pressure rose after exercise (see Table I) and in no case did it fall to nearly zero. Of the 8 cases showing a fall in diastolic pressure the greatest drop was 20 mm., the lowest 5 mm., and the average 10 mm. The test is sometimes useful, but a rise in the diastolic pressure after exercise does not invalidate the diagnosis of a patent ductus arteriosus.

THE ELECTROCARDIOGRAM

Brody and Randell (1935), Bullock *et al.* (1940), Touroff and Vessell (1940) and Holmes 1945) have all reported cases of mild left axis deviation. Many other changes have been reported but similar changes in the cardiogram were found in normal children by Lincoln and Nicholson (1928) and by Perry (1931). Gross (1940) pointed out that right axis deviation suggests the presence of some associated abnormality particularly pulmonary stenosis. Steinberg *et al.* (1943), among their 27 cases had 4 with left and 8 with right axis deviation. Tubbs (1945) said slight axis deviation was occasionally present. Gilchrist (1945) found left axis

deviation in 7 of his 13 cases all being below the age of 7 years. East (1945) found the cardiogram normal in all his 13 reported cases. In none of these series are the criteria of normality laid down. The general opinion appears to be that apart from mild left axis deviation the electrocardiograms are normal in this condition.

In the present series the axis deviation index as described by Paul White (1944) was measured. Cardiograms of 40 cases were available and only 2 cases showed axis deviation, there being one example of right and another of left deviation. No other abnormalities were present.

X-ray Examination

Post-mortem examinations have shown that the pulmonary artery, and either or both ventricles are frequently enlarged. Eppinger and Burwell (1940) X-rayed 9 cases and found the left ventricle was enlarged in 8, the pulmonary artery prominent in 8, and that pulmonary congestion was present in 9. Gross (1940) found the cardiothoracic ratio increased slightly, the pulmonary artery prominent and the lungs engorged. This was confirmed by Donovan *et al.* (1943) who found that among 50 cases most hearts were enlarged and the lungs congested. Grier (1943), and Steinberg *et al.* (1943) agreed. Shapiro (1944) in 62 cases found the heart enlarged in 40 and markedly enlarged in 4 of these. Gilchrist (1945) found 7 of his 27 cases did not have enlarged hearts on X-ray examination.

In the present series the normal ratio of the transverse diameter of the heart to the transverse diameter of the chest is taken to be 0·45 in adults and 0·52 in children with a normal variation of 10 per cent. Satisfactory X-rays were available in 15; the bounds of normal were exceeded in only one and the ratio in this was 0·66. All 15 had prominent pulmonary arteries. The degree of pulmonary congestion was measured by assessing the heaviness of the shadows around the hilum, but the incidence cannot be compared accurately as the variations of the normal are assessed differently by each observer. Of the 30 patients with X-rays suitable for examination, 15 showed congestion and the remainder were normal. In Group A, 8 of 20 cases showed congestion and in Group B, 7 of 10 cases are congested. This suggests that among an unbiased series of cases of patent ductus arteriosus (as opposed to a series containing many with symptoms) increased size of the heart and pulmonary congestion is less common than has so far been reported.

No correlation between the presence of symptoms and the presence of pulmonary congestion was found (Table II).

TABLE II

The Incidence of Symptoms

	Number of cases with symptoms	Number of cases with no symptoms	Total
Pulmonary congestion present ..	4	11	15
Pulmonary congestion absent ..	2	13	15
Total	6	24	30

Absence of the Machinery Murmur

Luys (1855) in 1848 saw a woman of 52 years in whom physical examination revealed no murmur; post-mortem the ductus arteriosus was patent. Duroziez (1862, 1863) found a woman aged 40 years with early congestive heart failure in whose chest he could hear no cardiac murmurs: the ductus arteriosus was patent, post-mortem. Foulis (1884) had a woman patient of 22 years with the typical basal systolic and diastolic murmurs in whom the murmur disappeared 25 days before she died of bacterial endocarditis. Balfour (1898) described a

cyanosed and breathless woman, who had a loud systolic murmur over the heart with a thrill over the second and third intercostal spaces to the left of the sternum; fifteen years later the murmur and thrill had disappeared: proof that this was a case of patent ductus arteriosus is hardly complete. Gaylor (1918) described a woman first seen by Babcock in 1898, who had a machinery murmur and thrill at the base of the heart; as the years went by the murmur vanished and post-mortem there was a saccular aneurysm of the aorta and a patent ductus arteriosus.

Gross (1939) said that in some children the ductus closed spontaneously and the diastolic element in the machinery murmur was lost. Shapiro and Keys (1943) described a woman of 46 years in heart failure over whose præcordium they could hear no murmur but post-mortem the ductus was patent. In the same paper they mention having seen 2 cases with the typical murmur that later vanished. Gilchrist (1945) described a boy aged 5 years with the typical murmur which had vanished at 6 years. When first seen his X-ray picture was characteristic of a patent ductus arteriosus but 18 months later it was normal. Jager (1940) and Gibb (1941) each described a woman, aged 55 and 51 respectively, with subacute bacterial endocarditis with no abnormal murmurs: post-mortem, the ductus was blocked in each case by vegetations.

The explanation of these findings appears to vary. Some may be explained by blocking of the ductus by vegetations in cases with infective endocarditis. The X-ray changes in Gilchrist's case appear to be due to actual closure of the duct. Other cases appear to be explained by the onset of heart failure. Some may be explained by thickening of the chest wall, emphysema, increase of adipose tissue, change in hæmodynamics, or in the position of the ductus relative to the chest wall. Gebauer (1943) found at operation that the size of the ductus and the amount of blood going through it varied as the intensity of the physical signs.

In the series under examination, 2 had well established machinery murmurs that have now vanished: neither case ever had a thrill. The first had a typical murmur on repeated examination between the ages of 8 and 13 years, a faint systolic murmur only could be heard at 15 years, and later at 21 years, and now at 25 years no murmur is audible. The second case was first diagnosed at the age of 10 years, a soft basal systolic murmur was heard at 24, and now 3 years later no murmur ean be heard. An X-ray of the first case at 9 years revealed slight pulmonary congestion and the cardio-thoracic ratio was 102/212=0·48. The pulmonary artery index, being the distance of the junction of the superior vena cava and the right auricle from the summit of the pulmonary arc, as described by Evans (1943), was 5·2 cm. At the age of 25, X-rays still show some pulmonary congestion, the cardio-thoracic ration is 117/263=0·44, and the pulmonary artery index is 7·1 cm. Evans (1943) found the index above 7·0 cm. in only 2 of 52 normal cases. The heart did not become smaller as described by Humphreys (1942) and others after operation. These findings suggest that the ductus is probably still patent. In the second case X-rays are not available. The blood pressure of the first case is 120/80, which does not indicate a leak but does not disprove the diagnosis. The second case has a blood pressure of 120/60, which is unaltered by exercise. Gross (1940) stated that closing the ductus by surgery causes the diastolic blood pressure to rise to normal. This reading, therefore, suggests that the ductus is still patent. Neither patient is obese or emphysematous. No definite conclusions can be drawn, but the findings suggest that in both the ductus arteriosus is still patent.

Physique

Abbott (1929) and Gross (1940) found their cases of patent ductus were usually of poor physique. In his 62 cases Shapiro (1944) found 32 were normally developed, 23 undersized, and 6 were obese. Gilchrist (1945) in 13 children found the physique above average in 2, average in 7, and below average in 4.

The height and weight of the present series has been compared with those of healthy children from the same area. The controls were chosen at random from schools scattered all over

TABLE III

A COMPARISON OF THE HEIGHTS AND WEIGHTS OF CHILDREN HAVING A PATENT DUCTUS ARTERIOSUS WITH NORMAL CONTROLS

Cases of patent ductus arteriosus				Normal Bristol children		Difference of case from controls	
Group	Age in years and sex	Height in cm.	Weight in kg.	Mean height in cm.	Mean weight in kg.	Height in cm.	Weight in kg.
A	3 F	106·0	14·5	94·4	15·4	+11·6	−0·9
A	3 F	98·0	14·1	94·4	15·4	+3·6	−1·3
A	4 F	116·0	17·7	103·5	16·5	+12·5	+1·2
A	4 F	109·5	15·9	103·5	16·5	+6·0	−0·6
A	6 F	111·0	20·5	117·4	21·9	−6·4	−1·4
A	8 M	131·5	25·0	128·5	26·7	+3·0	−1·7
A	9 F	—	27·3	—	29·0	—	−1·7
A	9 F	130·5	25·0	133·1	29·0	−2·6	−4·0
A	10 F	—	30·9	—	31·6	—	−0·7
A	12 F	—	27·3	—	40·4	—	−13·1
A	12 M	—	33·2	—	37·5	—	−4·3
A	13 M	—	29·1	—	45·0	—	−15·9
A	13 F	—	28·2	—	43·4	—	−15·2
A	15 F	—	50·0	—	52·5	—	−2·5
A	15 M	178·0	56·4	169·5	57·2	+8·5	−0·8
A	16 F	160·0	57·7	165·9	55·4	−5·9	+2·3
A	17 F	170·0	41·8	169·3	58·7	+0·7	−16·9
B	9 M	134·0	24·5	130·5	28·5	+3·3	−4·0
B	10 F	127·5	20·5	137·5	31·6	−10·0	−11·1
B	10 F	147·0	47·3	137·5	31·6	+9·5	+15·7
B	11 F	142·0	25·9	142·7	35·4	−1·8	−9·5
B	11 F	—	38·2	—	35·4	—	+2·8
B	14 M	156·0	42·3	166·2	50·0	−10·2	−7·7
B	17 F	—	54·5	—	58·7	—	−4·2

Bristol (Table III). For each year of age from 3 to 15, 50 children of each sex were taken and their height and weight measured. For children of 16 and 17 years only 26 and 10 controls were obtained. The figures for adults are compared with the figures for males and females aged 18, 20, and 25 years given by Cruickshank (1946) (Table IV). For these no statistical analysis has been attempted as there is no adequate series of controls.

The weights and heights of Group A have been analysed statistically because it is believed this group gives a true picture of the condition. The results have not been compared with Group B as there are not enough cases in this group with adequate data.

Weights are available for 17 children in Group A and 15 of these children were below the mean weight of control children of the same age and sex. An analysis of variance was made to determine the significance of the difference between the weights of the children in Group A and the corresponding normal children, i.e. of the same sex and age. It was found that on comparing the 4 abnormal males with the 4 classes of normal males of the same age the significance of the lesser weight in the Group A cases was expressed by $P<\cdot05$, i.e. was significant to the usual agreed 5 per cent level. In the case of the 13 females in Group A the significance was overwhelming, being expressed by $P<\cdot001$.

Heights are available for 10 children in Group A. Seven cases were above average height for their age and sex, while 3 were below the average. A similar analysis as for weights was made. In the two males the difference in height from the normal was not significant. In the 8 females the significance was expressed by $P<\cdot05$, i.e. the Group A cases are taller than the normals.

PROGNOSIS

Abbott (1936) found that of 92 uncomplicated cases of patent ductus examined post-mortem 43 per cent died of heart failure and 30 per cent of subacute bacterial endocarditis. Among

TABLE IV

A Comparison of Adults having a Patent Ductus Arteriosus with Normal Controls

Cases of patent ductus arteriosus				Normal adults (from Cruickshank)		Difference of case from normal	
Group	Age and sex	Height in cm.	Weight in kg.	Height in cm.	Weight in kg.	Height in cm.	Weight in kg.
A	20 F	160·0	58·6	167·7	57·7	−7·7	+0·9
A	22 F	162·5	58·6	167·7	57·7	−5·2	+0·9
A	23 F	162·5	52·7	167·7	57·7	−5·2	−5·0
A	25 F	172·9	55·9	167·7	59·1	+5·2	−3·2
A	25 M	175·4	62·3	175·4	63·6	0	−1·3
A	25 F	152·2	42·3	167·7	59·1	−15·5	−16·8
A	27 M	167·7	59·1	175·4	70·0	−7·7	−10·9
A	27 M	172·9	65·9	175·4	70·0	−2·5	−4·1
A	31 F	162·5	50·0	167·7	59·1	−5·2	−9·1
B	33 F	160·0	45·9	167·7	59·1	−7·7	−13·2

her 73 cases over 3 years of age 16 per cent died of bacterial endocarditis and 32 per cent of heart failure. In 60 collected cases Shapiro and Keys (1943) found the average age at death of men was 39 years and of women 35 years, i.e. 23 and 28 years respectively less than the normal expectation: 40 per cent died of bacterial endocarditis and 30 per cent of congestive heart failure. Sellors (1945) is even more gloomy and says the average expectation of life is 20 to 25 years. Wilson and Lubschez (1942) followed 38 cases for 20 years and in that time saw no deaths from bacterial endocarditis or congestive heart failure.

In Group A, 29 of the 30 cases were seen before the age of 15 years: the one exception was 23 years old when first seen and had been referred from an ante-natal clinic. At present the youngest person in the series is nearly 4 years old and the oldest 31 years. These cases have been followed for an average of more than 8 years and the average age when last seen was over 16 years. Of the cases in Group B, 9 were first seen before 15 years and 7 at a later age. The youngest in this series is now 7 and the oldest 47 years. These cases have been followed for an average of 8 years. Of these 16 cases 12 are still alive and their average age is 19 years. For the length of time the cases have been followed see Table V.

TABLE V

The Length of Follow Up of Cases of Patent Ductus Arteriosus

Time followed in years	Group A	Group B
0–5	9	4
5–10	7	4
10–15	6	2
15–20	7	0
20–	0	1

During the period under observation none of the Group A cases died or developed bacterial endocarditis or congestive heart failure. Among the 16 in Group B 1 case was first seen after recovery from bacterial endocarditis and 4 died of this disease; 1 of these 4 developed bacterial endocarditis and was treated with penicillin with apparent success although the blood sedimentation rate remained high. She returned home, but two months later suddenly developed fits and died the same night, presumably from a cerebral embolism due to a still active bacterial endocarditis.

The incidence of bacterial endocarditis (5 cases out of 16) in Group B is high because several were referred to hospital because of its presence, and this same reason must explain the high

incidence of bacterial endocarditis in published figures. The absence of such cases in Group A is striking and suggests that the frequency of bacterial endocarditis among all cases of patent ductus arteriosus must be lower than has so far been accepted. The true incidence must lie between that in the two Groups, but much nearer to that of Group A. The absence of congestive heart failure is not so significant as only a few of the cases have reached an age at which such a complication is likely.

The Indications for Surgery

Hubbard, Emerson, and Green (1939) gave retardation of growth, signs of aortic regurgitation, and cardiac insufficiency as the indications for surgery. Gross (1939) after 50 operations gave as his criteria, poor physique, cardiac embarrassment, bacterial endocarditis, and prevention of bacterial endocarditis. Marvin (1941), Burch (1944), and Tubbs (1945) gave almost the same criteria.

Gross (1940) said that operation caused the physical signs to vanish, the diastolic blood pressure to rise, the activity of the heart to diminish, the weight to increase, the heart size to decrease, and symptoms of heart failure to disappear. Donovan *et al.* (1943) found on X-ray that the heart size diminished only slightly, if at all, after operation. Hunter (1945) had 12 cases all of which lost their breathlessness and became mentally brighter after operation. Gilchrist (1945) found diminished lung vascularity and reduction in size of the heart in 14 patients who survived operation.

Gross (1943) had 2 deaths in 50 operations. Shapiro (1943) collected 104 reported cases treated surgically, none of which had bacterial endocarditis and found 9 deaths; presumably some of these were taken from Gross's records mentioned above. Gilchrist (1945) had 16 cases operated on with 2 deaths.

The absence of bacterial endocarditis in Group A suggests very strongly that it is much less common than has been described. Most cases lead a normal healthy life. On the other hand many are below average weight. Bacterial endocarditis has become much less dangerous since the discovery of penicillin and the facts published in this paper suggest that operation with its grave risks hardly seems justified merely for the prevention of the possibility of infection. However, cases cured of bacterial endocarditis appear to be proper subjects for operation as they are probably more liable to, or more exposed to infection. The risks of dental extraction or operation on infected tonsils are great but they can probably be almost obviated by cover with penicillin. The operative mortality is still high—possibly as high as, or higher than, the risk of bacterial endocarditis or congestive heart failure.

This investigation suggests that at present operation should only be undertaken in a limited number of cases. The indications for surgery would appear to be: bacterial endocarditis, recovered bacterial endocarditis, cardiac embarrassment, or poor physique.

A similar series of cases will need watching for at least 30 years before a complete answer can be given. It would be very worth while if the numbers were large.

Summary

A series of 46 cases of patent ductus arteriosus is described, particular attention being paid to 30 cases referred from a school cardiac clinic as these form a representative sample of the condition.

Females predominate over males by more than two to one.

Symptoms are few and rarely serious.

In two cases the classical murmur disappeared without other evidence that the ductus arteriosus had closed. Præcordial thrills sometimes vanish with increasing age. The diastolic blood pressure is usually normal; in only a limited number of cases does it fall after exercise. The electrocardiogram is normal.

Pulmonary congestion is not so common as has been reported. The average body weight is

significantly below normal. Bacterial endocarditis did not develop among the 30 cases referred from the school cardiac clinic.

Fewer cases should be operated on and the suggested indications for surgery are given.

I wish to acknowledge my indebtedness to Professor C. Bruce Perry without whose help this paper could not have been written, as he allowed me full use of his records and gave me much encouragement and advice; to Mr. H. Todd for great help with statistics; and to Professor Parry, Medical Officer of Health for Bristol, who supplied me with the heights and weights of normal children.

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CARDIAC CATHETERIZATION IN CASES OF PATENT INTERAURICULAR SEPTUM, PRIMARY PULMONARY HYPERTENSION, FALLOT'S TETRALOGY, AND PULMONARY STENOSIS

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The clinical and radiological findings in auricular and ventricular septal defects are well known. In many cases, however, the diagnosis may remain uncertain without pathological examination. The development of the technique of cardiac catheterization as a simple and safe procedure indicated a method for the detection of intracardiac shunts, and a number of reports on the use of the method have already been published (Brannon, Weens, and Warren, 1945; Dexter *et al.*, 1946; and Johnson, Wollin, and Ross, 1947). Not only can interauricular and interventricular shunts be demonstrated, but the amount of blood shunted and the pressure in the chambers of the right heart may be measured. In certain cases the demonstration that no shunt exists may be helpful in diagnosis. This paper reports data in a number of cases that were suspected on clinical grounds of having intracardiac shunts.

METHODS

The technique of cardiac catheterization and analysis of blood samples were similar to those previously published (McMichael and Sharpey-Schafer, 1944). Arterial samples were obtained from the brachial or femoral arteries. Average normal values are given in Fig. 1. The following procedure may be found convenient.

(1) A preliminary arterial sample indicates whether a right to left shunt has to be considered. We have often found it difficult to judge clinically whether cyanosis resulted from a diminished arterial oxygen saturation or a diminished peripheral flow.

(2) The catheter is passed into the right ventricle. In the absence of conditions causing increased cardiac output the existence of a left to right intracardiac shunt may be suspected if the sampled blood from this position appears bright red like arterial blood, and on analysis is shown to have a high oxygen content.

(3) The catheter is then withdrawn through the tricuspid valves into the right auricle and further samples taken. Brannon, Weens, and Warren (1945) who published data on four cases by similar methods, calculated the magnitude of the left to right shunt through an auricular septal defect from the gas content of auricular blood and the mean gas content of the S.V.C. and I.V.C. samples. With interatrial shunts auricular samples may not be completely mixed, and right ventricular samples are to be preferred if it is desired to make such calculations.

(4) Samples may then be obtained from the superior vena cava and, if possible, from the inferior vena cava.

In cases of right to left shunt the procedure should be carried out as quickly as possible with particular care to avoid clot formation on the tip of the catheter by a continuous drip

of heparinized saline, since there is a possibility of paradoxical embolism. Preliminary administration of heparin to the patient might be considered to lessen this risk. In children the investigation may be conveniently carried out under a basal anæsthetic.

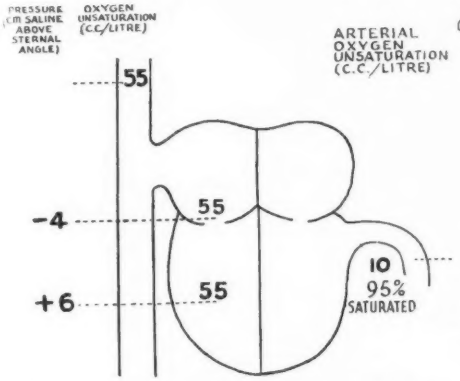

FIG. 1.—Average oxygen unsaturation levels and mean right auricular and ventricular pressure measurements in normal supine subjects.

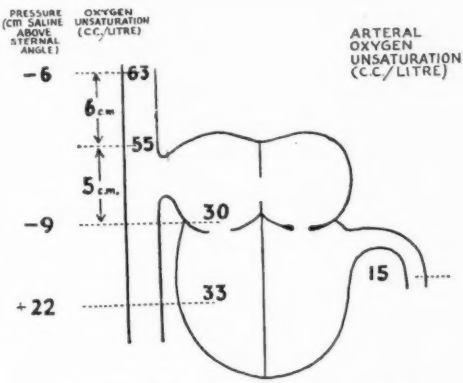

FIG. 2.—Case 1. Data obtained by cardiac catheterization.

CASE REPORTS

Case 1. A male, aged 57, was admitted complaining of loss of weight for two years and of bulky fatty stools for six months. An exploratory laparotomy had been performed elsewhere and a carcinoma of the head of the pancreas had been found. Slight dyspnœa on moderate exertion had been present for many years. There was no history of rheumatic fever. On examination there was no dyspnœa, cyanosis, or clubbing. The jugular venous pressure was normal. The blood pressure was 135/75 and the pulse regular at about 80 beats a minute. The heart was moderately enlarged and a systolic thrill and murmur were present, which were maximal at the apex. The hæmoglobin was 78 per cent. A cardiogram was not taken. Chest X-ray (Fig. 4, see p. 295) showed considerable enlargement of the pulmonary arteries, which pulsated strongly. The right ventricle and auricle were enlarged and the left auricle was not enlarged. The aortic knuckle was small. Total pancreatectomy was performed but the patient succumbed to post-operative complications.

Necropsy. The heart weight was 490 g. The pulmonary arteries were greatly enlarged. The right ventricle was thickened and dilated and the left ventricle was normal. The right auricle was enlarged and there was an interatrial septal defect (Fig. 3, see p. 294). The left auricle was normal in size. The mitral valve admitted two fingers and showed slight thickening of the free margins of the cusps.

Comment. The clinical findings suggested patent interauricular septum with possibly mitral incompetence. While catheterization (Fig. 2) clearly demonstrated the presence of a left to right interatrial shunt, the low normal right auricular pressure did not indicate any significant degree of mitral incompetence. Although the systolic thrill and murmur may have resulted from the slight thickening of the free margins of the mitral cusps, demonstrated post-mortem, they may also have been due to relative stenosis in the outflow tract from the right ventricle, in which chamber the mean pressure was considerably increased. During the prolonged and extensive operation for removal of the pancreas in this case, every effort was made to maintain right auricular pressure at or above the normal level by blood transfusion.

FIG. 3.—Case 1. Heart viewed from the left side to show auricular septal defect and slightly thickened mitral valve.

At one time right auricular pressure was as high as 7·5 cm. above the sternal angle; yet analysis of an arterial sample showed that there was no reversal of the shunt which remained from left to right auricle.

Case 2. A man, aged 20, came to hospital for treatment of acne. He had attended a special school until the age of 6, but there was no history of rheumatism. He played football and did not complain of breathlessness. The jugular venous pressure was not raised and no œdema was present. The blood pressure was 140/85. Auricular fibrillation was present, the apex rate being 120. Systolic and low-pitched diastolic murmurs were heard at the apex. The hæmoglobin was 94 per cent. An electrocardiogram showed auricular fibrillation, right axis shift, and slight depression of S–T III. Chest X-ray revealed a small aortic knuckle, enlargement of the pulmonary arteries, and no enlargement of the left auricle (Fig. 5, see p. 295).

Comment. A case of patent interauricular septum. The data (Fig. 6) showed that the shunt was from left to right. The diastolic murmur suggested that mitral stenosis may also have been present.

FIG. 4.—Case 1. Patent interauricular septum.

FIG. 5.—Case 2. Patent interauricular septum and mitral stenosis.

Case 3. A woman, aged 41, had been diagnosed as suffering from congenital heart disease as a child. She had had no cardiac symptoms until the age of 39, but during the three months preceding admission to hospital, dyspnœa had been increasing and there had been swelling of the ankles. The jugular venous pressure was raised to 6 cm. above the sternal angle, the liver was enlarged, and œdema was present. The blood pressure was 210/110. The heart rate was 80, with sinus rhythm. A systolic thrill and loud harsh systolic murmur were present, maximal in the third left interspace and conducted to the apex, and a short diastolic murmur was occasionally heard. The hæmoglobin was 89 per cent. The cardiogram showed no axis shift. There was slight depression of S–T in leads II and III, but all the T waves were upright. X-ray showed enlargement of both right and left ventricles, and moderate enlargement of the left auricle (Fig. 8, see p. 297). The pulmonary arteries were also enlarged.

Post-mortem examination confirmed the diagnosis of patent interauricular septum. The right ventricle was dilated and hypertrophied and there was moderate hypertrophy of the left ventricle. The mitral valve showed some small vegetations. There was cylindrical dilatation of the pulmonary arteries.

Comment. The data showed that the shunt was from left to right (Fig. 7), although right auricular pressure was considerably increased above normal.

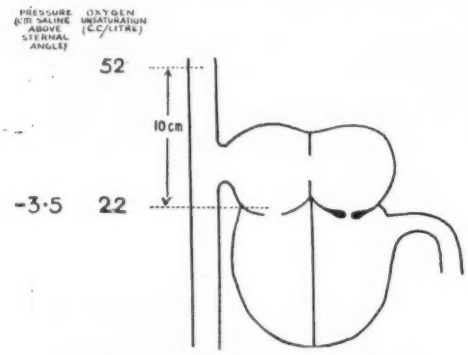

FIG. 6.—Case 2. Data obtained by cardiac catheterization.

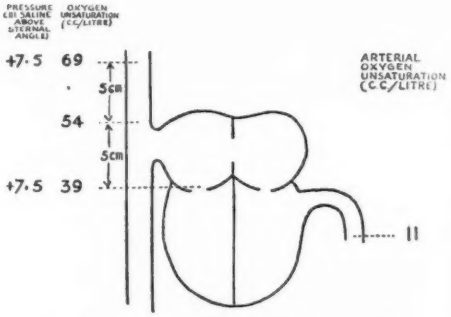

FIG. 7.—Case 3. Data obtained by cardiac catheterization.

Case 4. A woman, aged 40, gave a history of effort dyspnœa for 3–4 years and of attacks of nocturnal dyspnœa for 2 years. She had had rheumatic fever at the age of 11 years. The jugular venous pressure was not raised and there was no œdema. The blood pressure was 125/90. The heart rate was 108, with sinus rhythm. A harsh systolic murmur was heard over the lower part of the left side of the sternum, and the pulmonary second sound was increased. The hæmoglobin was 108 per cent. A cardiogram showed sinus rhythm, right axis shift and S–T II and S–T III depression. Chest X-ray showed enlargement of the right ventricle and of the pulmonary arteries (Fig. 9, see p. 297). The left auricle was not enlarged.

Comment. A case of patent interauricular septum. The data (Fig. 10) showed that the shunt was from the left to the right.

Case 5. A man, aged 31, had had two attacks of rheumatic fever 11 and 8 years previously. He complained of slight effort dyspnœa for 1 year. The jugular venous pressure was slightly raised. No œdema was present. The blood pressure was 105/70; the heart rate 70, and the rhythm regular. A systolic murmur was present at the apex and the pulmonary second sound was accentuated. The hæmoglobin was 118 per cent. A cardiogram showed right axis

FIG. 9.—Case 4. Patent interauricular septum.

FIG. 8.—Case 3. Patent interauricular septum.

shift with S–T III depression. X-ray examination showed enlargement of right and left ventricles with prominence of the conus: the left auricle was not enlarged and the aortic shadow small (Fig. 12, see p. 299).

Comment. The difference in oxygen content between superior vena caval and right auricular blood indicated a left to right auricular shunt (Fig. 11). The lower unsaturation figure obtained in the ventricle may have been due to inadequate mixing in the position of sampling in the auricle. The possibility of a small right to left ventricular shunt is suggested by the lowered arterial oxygen saturation.

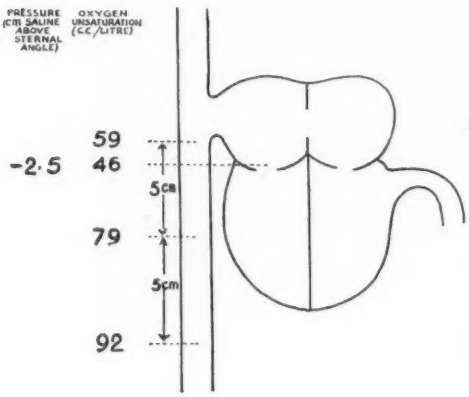

FIG. 10.—Case 4. Data obtained by cardiac catheterization.

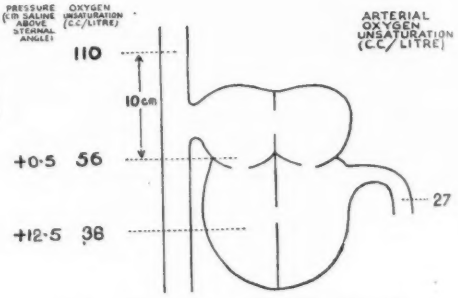

FIG. 11.—Case 5. Data obtained by cardiac catheterization.

Case 6. A woman, aged 26, had suffered from breathlessness and blueness of the lips on exertion for one year. There was no history of rheumatic fever. The jugular venous pressure was raised to 1 cm. above sternal angle level, but the liver was not enlarged and there was no œdema. The blood pressure was 114/76. There was a harsh systolic murmur at the apex and the pulmonary second sound was accentuated. A cardiogram showed sinus rhythm, right axis shift, and depression of S–T in leads II and III. P II was 0·3 mv. A chest X-ray showed enlargement of the right ventricle, the conus and the pulmonary arteries, but no enlargement of the left auricle (Fig. 13, see p. 299). The vital capacity was 2300 ml. She developed severe cardiac failure and cachexia with extreme peripheral cyanosis. Death occurred 18 months after the first admission to hospital.

Post-mortem examination. The heart weighed 481 g. and showed hypertrophy and dilatation of the right ventricle. The right auricle was dilated, but no septal defect was found. The left ventricle and auricle were normal, and so were the valves. Atheroma of the pulmonary arteries with some recent thrombosis was found, and the small branches of the pulmonary artery showed great thickening of the arterial wall and reduction of the size of the lumen. There was no pulmonary emphysema.

Comment. The samples from the right ventricle and auricle showed no evidence of a left to right shunt (Fig. 14). Arterial saturation was originally normal though slightly reduced 8 months later when severe failure was present (Fig. 15). There was no evidence of right to left shunt or of chronic disease in the lungs. These findings with the demonstration of a greatly raised mean right ventricular pressure suggested a diagnosis of primary pulmonary hypertension. The post-mortem findings supported this view.

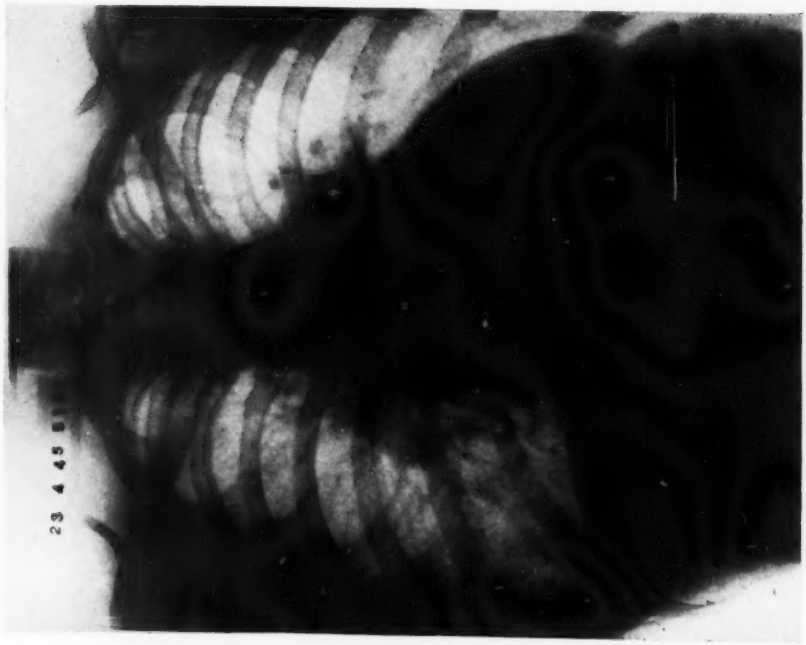

FIG. 13.—Case 6. Primary pulmonary hypertension.

FIG. 12.—Case 5. Patent interauricular septum.

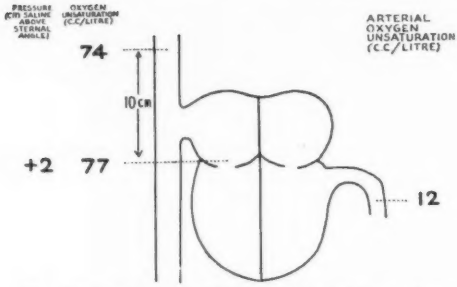

FIG. 14.—Case 6. Initial data obtained by cardiac catheterization.

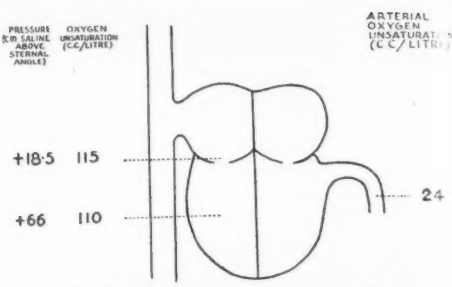

FIG. 15.—Case 6. Data obtained 8 months later when severe failure was present.

Case 7. A boy, aged 15, had been diagnosed at the age of 4 as a case of Fallot's tetralogy. He was admitted to hospital following hæmoptysis. He was cyanosed, showed defective growth, and had clubbing of the fingers. The jugular venous pressure was not increased, the liver was not enlarged and there was no œdema. The blood pressure was 80/20, and the heart rate 96, the rhythm being regular. A harsh systolic murmur was heard, maximal in the third left interspace. A cardiogram showed sinus rhythm, right axis shift and depression of S–T III. Chest X-ray showed a classical " sabot " heart (Fig. 18, see p. 301).

Comment. The decrease in the saturation of arterial blood indicated a considerable shunt from right to left (Fig. 16). The difference between right auricular and ventricular blood suggests the possibility that there was also some mixing from the left to the right ventricle.

Case 8. A girl, aged 8, was known to have had a congenital heart lesion since birth. In recent months she had become easily fatigued and after mild exertion the jugular venous pressure became elevated. On examination there was no clubbing or cyanosis. The jugular venous pressure was at the level of the sternal angle at rest and the liver was palpable. A systolic thrill and a harsh systolic murmur were present in the third left interspace, and the pulmonary second sound was accentuated. The blood pressure was 100/50. A cardiogram showed right preponderance. Chest X-ray showed enlargement of the pulmonary arteries and right ventricle (Fig. 19, see p. 301).

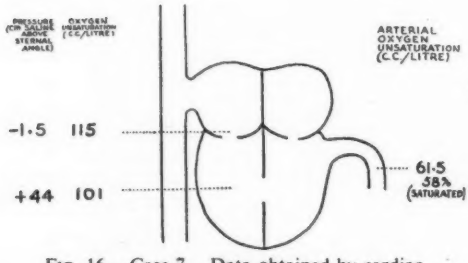

FIG. 16.—Case 7. Data obtained by cardiac catheterization.

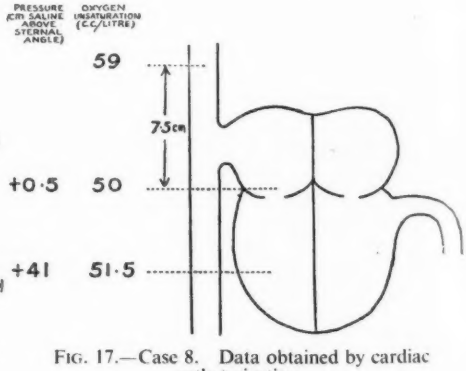

FIG. 17.—Case 8. Data obtained by cardiac catheterization.

Comment. In view of a pulse pressure of 50 mm. Hg. patent ductus was considered, while a left to right auricular shunt could not be excluded on clinical grounds. Catheterization

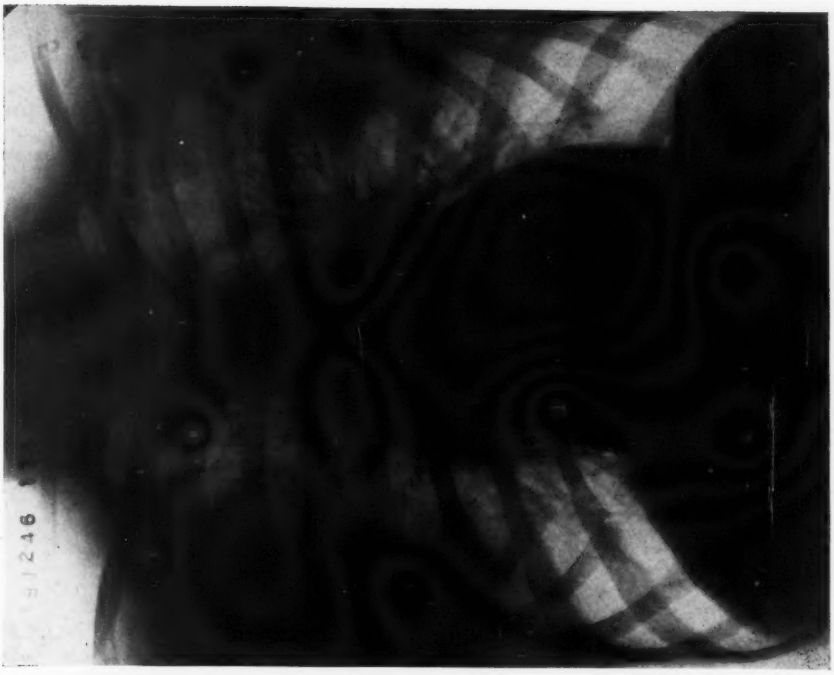

FIG. 19.—Case 8. Pulmonary stenosis.

FIG. 18.—Case 7. Fallot's tetralogy.

revealed no evidence of a shunt (Fig. 17). Although an attempt to pass the catheter into the pulmonary artery failed, the greatly increased right ventricular pressure made a diagnosis of pulmonary stenosis almost certain.

Case 9. A boy, aged 13, was known to have had cardiac murmurs since birth. He played games at school and there was no history of cyanosis or excessive breathlessness. The jugular venous pressure was not raised and there was no œdema. The blood pressure was 90/40 and the heart rate was regular at 90. A thrill, a harsh systolic murmur, and a soft diastolic murmur were present in the pulmonary area, maximal in the second left interspace. The pulmonary second sound was accentuated. A diastolic murmur was present at the apex. The hæmoglobin was 91 per cent. A cardiogram was normal. X-ray examination showed considerable enlargement of the right ventricle and the pulmonary arteries. The left ventricle was also slightly enlarged. There was no left auricular enlargement.

Comment. A case of patent ductus arteriosus (Fig. 20). The mean pulmonary arterial pressure was surprisingly high, in view of the systemic blood pressure. Considerable increase in right ventricular work from this high pressure may have resulted in hypertrophy comparable with the left ventricular hypertrophy from increased output, since the cardiogram shows no axis deviation.

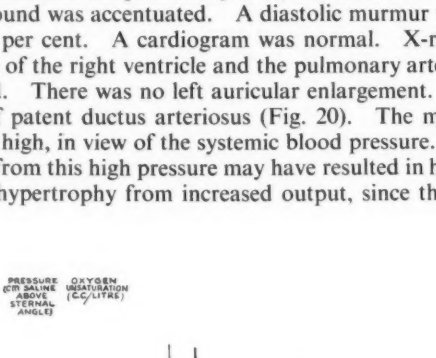

FIG. 20.

DISCUSSION

The precise diagnosis during life of congenital heart lesions has now more than an academic interest. Ligation of a patent ductus arteriosus is a well-established method of treatment, while the procedures of Blalock and Taussig (1945) in Fallot's tetralogy seem very promising. Even if the presence of a patent ductus is obvious, it may be of value to know whether other lesions are present or absent when considering ligation. Enlargement of the pulmonary arteries, as a main finding, has long presented a difficult differential diagnostic problem for clinicians and radiologists. Patent interauricular septum, mitral stenosis, pulmonary stenosis, Eisenmenger's complex, patent ductus arteriosus, cor pulmonale from emphysema, primary pulmonary hypertension and rupture of a syphilitic aorta into the pulmonary artery are among the conditions that may have to be considered. Investigation of the right heart by catheterization may provide the answer, or at least further data on which a firmer diagnosis may be based.

The conditions under which a left to right auricular shunt is reversed remain obscure. Like Brannon, Weens, and Warren, we have tried various procedures such as the Valsalva experiment and different postures without producing any change in the arterial oxygen saturation. The intrathoracic pressure rises steeply during a Valsalva, and all chambers of the heart may be affected equally. When the mitral valve is stenosed, any procedure tending to raise the pressure in the right auricle may result in at least an equal rise in the left so that the shunt remains left to right. Case 3 shows that mean pressure in the right auricle may be high and the shunt still left to right. In Case 1, where the mitral valve was probably functionally normal, a high right auricular pressure produced by transfusion did not reverse the shunt.

It is difficult to make an exact quantitative estimate of the proportion of blood shunted from the left to the right heart. Brannon, Weens, and Warren estimated that in some cases of patent interauricular septum the output of the right ventricle might be as much as three times that of the left. While the systemic blood flow may be estimated from vena caval samples, it is not so easy to get an exact estimate of the output of the right heart. The inflowing stream of arterial blood from the left auricle makes it difficult to be certain that samples from the right heart are completely " mixed." This difficulty is illustrated in Case 5.

The normal mean right ventricular pressure is from 10 to 15 cm. of saline above the mean auricular pressure. The high ventricular pressures recorded in Cases 6, 7, and 8 indicated increased resistance in either the outflow tract of the right ventricle or in the pulmonary arteries. In congestive heart failure from any cause, mean right ventricular pressure may be increased. This finding, therefore, is more significant of organic obstruction in the right outflow tract if right auricular pressure is normal.

Summary

Samples of blood from the right auricle and right ventricle may be obtained by cardiac catheterization in cases of suspected cardiac shunts. Blood so obtained contains more oxygen than vena caval blood if the shunt is from left to right. Right to left shunts may be detected by a reduced arterial oxygen saturation in the presence of normally functioning lungs.

Two of the five cases with patent interauricular septum showed a conspicuous increase in right auricular pressure with the shunt still left to right.

A case with enlarged pulmonary arteries and a normal arterial oxygen saturation showed no evidence of a shunt and a greatly increased mean right ventricular pressure. A diagnosis of pulmonary hypertension was confirmed post-mortem.

A case of Fallot's tetralogy showed an increased mean right ventricular pressure. The data suggested some left to right mixture of blood although the main shunt was from right to left.

A child with enlarged pulmonary arteries showed no evidence of a shunt. Mean right ventricular pressure was greatly increased and a diagnosis of pulmonary stenosis was made.

A case of patent ductus arteriosus showed a greatly raised mean pulmonary arterial pressure, a considerably raised right ventricular pressure, and highly oxygenated pulmonary arterial blood samples.

We are indebted to the Medical Research Council for a personal grant to one of us (S. H.) and for an expenses grant.

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PROCEEDINGS OF THE BRITISH CARDIAC SOCIETY

The ELEVENTH ANNUAL GENERAL MEETING of the British Cardiac Society was held at Guy's Hospital, London, on Tuesday, May 6, 1947. Chairman: MAURICE CAMPBELL. The Chairman took the chair at 9.30 a.m.; 62 members and 19 visitors were present.

PRIVATE BUSINESS

1. The minutes of the last meeting having been printed in the Journal (**8**, 233, 1946) were approved and signed.
2. The Secretary reported that a balance of £8 16*s*. 8*d*. had been handed over at the transfer of the Secretaryship and said that subscriptions amounting to £78 were due in 1947–48. It was decided that the subscription should be increased to 20*s*. for Ordinary members and 10*s*. for Associate members.
3. Boyd Campbell (Belfast) and McMichael (London) were elected members of the Council for the years 1947–51.
4. The following new members were elected:

Ordinary Members	*Associate Members*	
Wyn Jones	D. Rhodes Allison	R. W. Luxton
Lovibond	Roland E. Anderson	Wilfred Oliver
Sharpey-Schafer	Ronald Hartley	R. Kemball Price
Suzman	Ronald Jones	Richard Turner
Swan	Geoffrey Konstam	

Five Associate members were re-elected for a further period of three years.

5. The following changes in the Rules were carried, *nem. con*., on the recommendation of the Council:
   Rule 8. Substitute " Rules 12 and 22 " for " Rule 12 ".
   Rule 17. Add at the end " and shall take over office from his predecessor at the start of the meeting ".
   Rule 21. Insert before " the Chairman-Elect ", " the Chairman ".
6. The Secretary reported that as a result of representations to the President of the Royal College of Physicians, a Committee on Cardiology had been constituted by the College. The Report of this committee, dealing with the training of specialists in cardiology, the practice and teaching of cardiology, and the planning of cardiac services under the National Health Scheme, had been published and copies were available. The Secretary also announced the progress of arrangements for the International Conference of Physicians at which the Section of Cardiology would be under the Chairmanship of Sir Maurice Cassidy.
7. The Council of the Society was authorized to decide on the election of new Honorary Members after the conclusion of the International Conference of Physicians.
8. The Surgical Treatment of Hypertension was chosen as the main discussion at next year's meeting.

DISCUSSION ON CARDIAC EMERGENCIES

(1) ACUTE RIGHT AND LEFT HEART FAILURE: opened by BRAMWELL and MCMICHAEL.
(2) CARDIAC INFARCTION: opened by BOYD CAMPBELL and HILL.
(3) LOSS OF CONSCIOUSNESS: opened by MAURICE CAMPBELL and LENDRUM.
(4) PULMONARY EMBOLISM: opened by PAUL WOOD.

CRIGHTON BRAMWELL said that the two most important precipitating causes of *acute heart failure* were coronary occlusion and pulmonary embolism, both of which would be dealt with by later speakers. He also mentioned the possibility of rupture of a cusp of the

aortic valve and of sudden occlusion of the mitral orifice by a ball thrombus in the auricle. He then referred to two causes of acute heart failure that had received less general recognition. Sudden failure of the left auricle in association with severe mitral stenosis might occur unexpectedly, and he had reported two cases seen during pregnancy. Secondly acute heart failure due to vitamin B_1 deficiency, though rare in this country was important since the treatment was specific and lesser degrees of vitamin deficiency might be a contributing factor in the production of heart failure.

J. McMICHAEL divided his remarks into acute left and acute right heart failure.

Acute Left Heart Failure. Opportunities had occurred of observing attacks of acute orthopnœa with the cardiac catheter in situ. The arterial pressure rose above the previous level even in hypertensive subjects. The right auricular pressure invariably rose in the attack and, as the lungs became congested, presumably the left auricular pressure also rose considerably. Blood returning to the right heart contained less oxygen than before the attack, but owing to the increased oxygen uptake resulting from the struggle for breath the cardiac output was found to be slightly increased.

In *treatment* the most effective and reliable remedy seemed to be *morphine*, under the influence of which right auricular pressure, metabolic rate, and arterial pressure fell. It was not yet possible to say what was the exact sequence of events either in the precipitation of the attack or in the mechanism of its relief. After morphine the next most reliable remedy was *theophylline-ethylene-diamine* which often reduced the right auricular pressure within 5 minutes. *Venesection* should also be recognized as a most effective, though less convenient, measure. *Intravenous digoxin* may not be satisfactory in these acute seizures. An attack had actually been observed to come on following intravenous digoxin and perhaps precipitated by the pressor action of this drug on the arterial system.

Acute Right Heart Failure. The most striking instances occur in acute pulmonary embolism. This subject is dealt with by Paul Wood. Patients with emphysema heart, however, are liable to develop attacks of congestive failure during acute exacerbations of bronchitis. In these attacks the cardiac output is high and the raised venous pressure should be regarded as to some extent a compensating mechanism. Reduction of venous pressure by venesection or digoxin often brings down the cardiac output, and these measures are thus of little benefit as a rule (*Clin. Sci.*, 1947, **6**, 187). The best treatment at this stage is an oxygen tent to improve the oxygen content of the blood, and measures to overcome the infection.

S. B. BOYD CAMPBELL discussed the possible ætiology of *coronary infarction.* Case histories of 169 hospital and 100 private patients were investigated. Age groups, occupation, fat consumption, and previous infections were noted; 85 per cent of the private and 92 per cent of the hospital cases occurred in the 40 to 70 age group with 37 per cent and 31 per cent in the 50 to 59 group. Over-indulgence of fatty foods has been prevented for many years by rationing and seems to have no bearing on the increase in infarction. Occupation in both private and hospital cases showed similar findings. No special occupation seemed to be associated with the occurrence of infarction. Occupations found included civil servants, clergymen, clerks, farmers, skilled workmen, labourers, housewives etc. Stress and strain seemed to have no bearing on the ætiology though the actual onset in some cases was associated with exertion and in others followed a heavy meal.

It was difficult to find any definite infection in the history of previous illnesses. Some cases had had some feverish illness within a few weeks of the infarction. Case histories were of little help in solving the ætiology.

He was indebted to J. H. Biggart, M. G. Nelson, and Florence McKeown for the lantern slides illustrating their recent work on coronary artery disease. First slides were shown from

cases dying of acute rheumatism. The initial change was an acute arteritis affecting the media followed by intimal changes with healing fibrosis and then secondary degeneration. The intimal fibrosis may almost occlude a large coronary artery. Similar changes were shown in coronary arteries of rabbits injected with horse serum. The final stages was demonstrated in a series of lantern slides of sections taken from the site of the coronary thrombosis. Intramural intimal hæmorrhage with fibrinoid degeneration preceded the thrombosis and was shown to be the precipitating factor. Secondary atheromatous changes were demonstrated. While hypertension plays a part in initiating the intramural hæmorrhage there seems to be evidence that all the changes from the initial medial arteritis to the fibrinoid degeneration and intramural hæmorrhage are an allergic reaction in patients sensitized to some protein resulting from an infection or possibly ingested.

I. G. W. HILL spoke on the *electrocardiographic aspects of cardiac infarction.*

MAURICE CAMPBELL said that even if *loss of consciousness* is rarely an emergency in the sense of needing urgent treatment, the anxiety caused and the difficulties of diagnosis justify its inclusion. The same word, syncope, is used for trivial fainting attacks and for a method of death and should, therefore, be banished from scientific discussion. It might be useful if it was recognized that syncope meant a sudden loss and fainting a gradual loss of consciousness, and he had sometimes used the terms in this sense. There is, however, no such agreement and it would at present be wrong because syncope is by definition (O.E.D.) "a failure of the heart's action, resulting in loss of consciousness or sometimes in death."

A more accurate knowledge of attacks of unconsciousness will be difficult to acquire without more accurate descriptions and this should be helped by more precise definition of terms. The speed with which consciousness is lost seems one of the most fundamental distinctions; and I suggest that the word tachy-a-psychia should be used for the sudden loss of consciousness, seen in most Stokes-Adams attacks or in epilepsy, and brady-a-psychia for the gradual loss of consciousness seen in most fainting attacks in young people. Lipo-psychia is used most often by Hippocrates for swooning, but apsychia is an alternative that makes the compound words euphonious. I am indebted to Mr. Yorke, Classical Tutor at New College, for coining these words for me.

Loss of consciousness concerns the physiologist and neurologist even more than the cardiologist; but there is, perhaps, more knowledge of the provoking mechanism of unconsciousness in disorders of the heart than of the nervous system. The common factor in the former group is the production of cerebral anoxæmia—whether by changes of rhythm, including ventricular standstill as in many Stokes-Adams attacks; by standstill of the heart as in a few fainting attacks or during anæsthetics; by the combination of a slow heart and a fall of blood pressure or by one of these alone, as in most fainting attacks and in many cases of the carotid sinus syndrome; or by temporary arrest of the cerebral circulation by venous stasis or difficulty in auricular filling during violent spasms of coughing or after pulmonary embolism. Anoxæmia without a change in the blood flow must also be remembered.

Broadly speaking, these various cardiovascular causes—and there are many more—can be divided into those where there is arrest of the cerebral circulation, often but not necessarily with cardiac or ventricular standstill, and those where there is diminution of the cerebral circulation (as in the severe fall of blood pressure after a large cardiac infarct or with a very rapid paroxysmal tachycardia); this may reach a stage of deficiency severe enough to cause unconsciousness which will therefore generally be gradual.

As a rule, therefore, tachyapsychia will mean sudden arrest of the cerebral circulation and bradyapsychia a gradual diminution of the cerebral circulation: there are, however, exceptions and, of course, many cases of tachyapsychia will be cerebral in origin. Arrest of the circulation

has greater dangers of the heart failing to start again and so tachyapsychia often has a much graver prognosis than bradyapsychia.

Generally, however, a patient who has been unconscious can give the physician no direct indication as to whether the cause was cardiac or cerebral and the decision may be very difficult and may call for much knowledge and experience: the symptoms produced in either way may be almost identical. It is, in particular, essential to be on familiar terms with epilepsy, idiopathic or symptomatic, in both its major and minor forms,

The duration of the attack is of great importance in this connection. A short attack, and this means one of not much more than two minutes, may be either cardiac or cerebral in origin. A long loss of consciousness, and this means one lasting more than 5 or 10 minutes must be wholly or partly cerebral—partly, when a short cardiac arrest started the loss and produced changes in the cerebral cortex that prolonged the unconsciousness.

I shall make some rather dogmatic statements that will help to clarify the discussion. Arrest of the cerebral circulation for from 5–10 seconds by a pressure cuff round the neck (Rossen, Kabat, and Anderson, *Arch. Neurol. Psychiat.*, 1943, **50**, 510) causes sudden loss of consciousness. Standstill of the heart or of the ventricle for from 5–10 seconds also causes sudden loss of consciousness, so this is further evidence that both act in the same way by producing sudden arrest of the cerebral circulation.

In unconsciousness due to cerebral anæmia, spasmodic movements or fits are dependent on the depth and duration of unconsciousness and not on its cause; and may, therefore, occur in attacks of all sorts. They follow 15–20 seconds after the arrest of the circulation.

Arrest of the cerebral circulation for 2 minutes in experiments on men or arrest of the circulation for 2 minutes by standstill of the heart in disease or during anæsthetics is compatible with perfect recovery.

Standstill of the heart for 5 minutes is generally permanent, i.e. the patient is dead. In the rare cases where the heart starts again after a longer interval than this, anoxæmia will have caused irreversible damage to the brain, especially to the cortex. The first sign of this cerebral damage will often be a much longer period of unconsciousness. Where the cerebral arteries are diseased as in many cardiac cases and most Stokes-Adams attacks a shorter period than 5 minutes may produce cerebral damage from which it is not possible to make a recovery.

Standstill of the heart for some time more than 2 minutes but less than 5 minutes will produce cerebral damage from which it may be possible to make a perfect recovery. The first sign of this cerebral damage may be a more prolonged period of unconsciousness. This means that even in a case known to be cardiac and known to be unconscious for many minutes, it is of great prognostic significance to know when the circulation returns, i.e. when the heart starts beating. Ultimately this period of between 2 and 5 minutes may be defined more precisely though there will always be great danger about recovery in such cases. In clinical records of heart standstill periods of from 20 to 80 seconds are not often exceeded and these are well within the limits when complete recovery can take place.

R. G. LENDRUM mentioned some of the conditions where a cardiac ætiology is wrongly and not infrequently diagnosed. Cerebro-vascular epilepsy and even hypertensive crises can thus be mistaken, especially where the patient is already under care for cardiovascular disease. Another is idiopathic epilepsy, if beginning in middle age, showing little or no movements and possibly having a prolonged sensory aura. Epilepsy of the chronic alcoholic is aided in joining this group by wrong assessment of the cyanotic complexion.

Patients who take attacks of hysterical hyperventilation with " unconsciousness " are looked on as liable to " heart attacks," this being most likely where the process starts with retrosternal discomfort (globus hystericus).

In nervous, hypersensitive people, consciousness of even a single ventricular extrasystole

gives rise to faintness and fear that consciousness will be lost. Actual appreciation of the feeling of " falling over to sleep " can occur. Here too a similar acute fear arises and in both cases the resulting tachycardia and palpitation may lead to diagnostic confusion.

Faintness after great unaccustomed effort occurs in healthy people and the patient and even his doctor, may decide that the heart has been " strained."

Aerogastric bloquée gives discomfort in the chest, faintness, and a premonition of death. This is most likely in someone already affected with cardiac disease and thus is all the more readily diagnosed as a " heart attack."

PAUL WOOD analysed twenty cases of massive *pulmonary embolism*. Of the clinical features special attention was directed to the frequency of substernal pain (12). Detailed studies of the behaviour of the arterial and venous blood pressure were presented. The arterial pressure fell abruptly and profoundly, reaching its lowest level within a matter of minutes or hours, and thus differed from its behaviour in acute myocardial infarction. The four patients with the lowest pre-embolic blood pressures all died at the time of the profound fall; no others died. The venous blood pressure rose sharply in all but two cases. A graph based on daily readings showed that the maximum elevation occurred within the first 24 hours, and was followed by a steady decline towards normal which was reached in 3–7 days. Maximum levels ranged between 2 and 10 cm. above the sternal angle.

The relationship of the venous blood pressure to the cardiac output was studied in one case. When the venous pressure was lowered by means of cuffs on the thighs, the cardiac output fell. When the cuffs were removed the venous pressure and the cardiac output rose. It was concluded that the raised venous pressure was beneficial and that the heart was not overloaded. The right ventricular pressure in this case, measured by means of cardiac catheterization, was +32 cm. of saline above the sternal angle with the subject nearly horizontal.

The rest of the communication dealt with the electrocardiographic diagnosis. The multiple chest lead pattern previously described by the author (*Brit. Heart J.*, 1939, **1**, 49, and 1941, 3, 21) was further amplified. The changing pattern was attributed to transient right ventricular stress, and was met in other conditions besides massive pulmonary embolism.

SHORT COMMUNICATIONS

CONSTRICTIVE PERICARDITIS

H. COOKSON

Three cases were reported illustrating variations in the onset and in the course of chronic constrictive pericarditis. In the first, a man of 24, the onset was acute with pericardial effusion and six months later the full picture of chronic constrictive pericarditis had developed. Pericardectomy was done one year after the onset, and was partially successful, ascites persisting; because of technical difficulties adequate resection was not possible. The second case, a woman of 22, had an insidious onset with shortness of breath, puffiness of the face and swelling of the legs; there was X-ray evidence of old pleurisy. While under observation ascites developed and there was recurrent bilateral dry pleurisy. Pericardectomy was done 16 months after the onset; result good. The third case, a girl of 16, had abdominal symptoms at the onset. Over a period of 33 months the spleen has gradually enlarged and now fills more than half the abdomen. Gross thickening of pleura on both sides; extensive calcification in the pericardium; short attacks of paroxysmal tachycardia (auricular). The signs of pericardial constriction are moderate and because of this and because of the huge spleen, of which the cause is obscure, pericardectomy has not yet been undertaken.

There was no bacteriological proof of tuberculosis in any of the three cases. Cardiograms were very similar, showing S–T depression and T inversion. In the operated cases pulmonary

shadows, presumably due to pleural thickening, diminished. Little change in cardiac outline occurred after pericardectomy, but the abnormalities of T and S–T diminished.

Continuous Recording of Systolic Blood Pressure during the Unconsciousness of Sudden Anoxia

F. Latham. (Introduced by W. K. Stewart.)

A method of continuous systolic blood pressure recording as employed at the R.A.F. Institute of Aviation Medicine has been described, and also its application to the study of the unconsciousness of sudden anoxia.

The technique utilizes pulse waves passing under an arm cuff to actuate a solenoid valve which automatically maintains the pressure in the cuff at systolic blood pressure level. The pulse waves are detected by means of a photoelectric cell applied to the finger.

During a preliminary series of experiments, subjects were exposed in the decompression chamber to simulated altitudes of 35,000–40,000 feet and continuous blood pressure records taken during the subsequent anoxia, which ensued when their oxygen was cut off. The subjects were given oxygen when they became unconscious and the duration of each anoxia run was between 30–90 seconds. Difficulty was encountered in some of the experiments owing to convulsions occurring prior to unconsciousness.

In the four cases investigated at this stage there did not appear to be any evidence of cardiovascular collapse and the blood pressure remained elevated throughout.

A Case of Long-Standing Auricular Fibrillation

By Crighton Bramwell. To be published in full.

Familial Cardiomegaly

By William Evans. To be published in full.

Peripheral and Central Venous Pressures in Tricuspid Incompetence

By E. P. Sharpey-Schafer. To be published in full.

Right Auricular Dilatation Without Valve Disease

J. L. Lovibond described the case of a man of 28 who for the past 4 years had suffered from symptoms of chronic right heart failure with swelling of the neck, abdomen, and legs. An intracardiac thrombus was diagnosed during life but the underlying pathology was not revealed until necropsy. There was no history of rheumatic or other infection but, at the age of 14, his heart had been questioned because of cyanosis on swimming. At 21 tonsillectomy was performed for sore throats. At 24 his abdomen began to swell and he was found to have ascites, a big liver and spleen, radiological enlargement of the right auricle and superior vena cava, and auricular fibrillation. Failure was treated appropriately and normal rhythm restored by quinidine. From now on he became increasingly short-winded and ascitic, requiring regular abdominal paracenteses. For most of the next 4 years he remained in hospital. At 26 he had a lung infarct and later in the year underwent omentopexy without relief. At 27 venous pulsation in the neck and a striking apical triple rhythm were noted, but no murmurs. There was sinus rhythm with wide, bifid P waves in leads II and III and right axis deviation. There was no response to diuretics and his abdomen now required fortnightly tapping. Although a few months later he reverted to fibrillation and also suffered a further lung infarct his general condition continued to be well maintained. The absence of clinical evidence of tricuspid or other valvular disease and the exclusion of cardiac tamponade by aspirations of a small pericardial effusion without beneficial effect, together with the history of lung infarcts and the radiological finding of a dense inert shadow in the position of the right auricular appendix, prompted the diagnosis of right auricular thrombus. In the face

of constant requests for surgical relief from the patient, an intelligent salesman, operation was eventually agreed to and an adherent laminated thrombus (6·2×5·0×1·2 cm.) was removed from a tensely dilated right auricle. Death occurred a few minutes later from pulmonary embolus. The heart (500 g.) showed great dilatation and hypertrophy of the right auricle and moderate hypertrophy of the left, but the ventricles, valves, aorta, coronary and pulmonary arteries were normal. Microscopically there was diffuse myocardial fibrosis with great muscle hypertrophy and focal endocardial thickening of both auricles and, to a lesser degree, both ventricles. The liver (1485 g.) and spleen showed "sugar icing" capsular thickening with advanced congestive reticular fibrosis of chronic back pressure. The pericardium and peritoneum were unaffected, and the lungs normal apart from old and recent infarcts. The condition was regarded as a healed generalized myocarditis of unknown origin, affecting the whole heart but principally the right auricle, causing its inordinate dilatation with resulting back pressure effects. The resemblance of the cardiac pathology to Fiedler's isolated myocarditis and to cases described by Kugel (1939), Levy and von Glahn (1944) and Bedford and Konstam (1946) were briefly discussed. A second case was shown of aneurysmal right auricle with fibrosis and focal stretching of its wall, due to thrombotic occlusion of the right auricular branches of the coronary artery.

A New Sign in Coarctation of the Aorta

S. Suzman demonstrated a new sign in coarctation of the aorta by means of which the superficial collateral arteries of the back and sides of the chest are caused to become visible when not visible previously, or to become much more prominent. The sign is best produced by causing the patient to stoop or bend forward and with the arms hanging down vertically. An equally good method is for the patient to lie prone on a couch with a pillow under the chest and the arms hanging down over the edge of the couch. A good light is advantageous. The explanation for this phenomenon is that there is partial compression and obstruction (in cases where the sign is positive) of the subclavian artery in the costo-clavicular space, and any change of posture that widens this space will thus release pressure on the subclavian artery and so cause a sudden filling up of superficial arteries so that they suddenly appear in places where none were visible before or become more prominent.

In three cases out of four so tested, the sign was positive (subsequently three more cases tested showed this sign). He suggested that this sign should be looked for as a routine in all cases of suspected coarctation of the aorta and in fact all cases of high blood pressure, for by this means there might be less likelihood of missing a superficial collateral circulation.

The Retinal Vessels in Hypertension

By A. Leatham. (Introduced by William Evans.) To be published in full.

Partial Superior Vena Caval Block

By Sheila Howarth. (Introduced by Sharpey-Schafer.) To be published in full.

Mass Thrombus of the Left Auricle

By R. Benson. (Introduced by William Evans.) To be published in full.

Lone Auricular Fibrillation

By S. Prichard. (Introduced by William Evans.) To be published in full.

PROCEEDINGS OF THE INTERNATIONAL CONFERENCE OF PHYSICIANS

PRESIDENT: THE LORD MORAN, M.C., M.D., P.R.C.P.

SECTION OF CARDIOLOGY

PRESIDENT: SIR MAURICE CASSIDY, K.C.V.O., C.B., M.D., F.R.C.P.

The INTERNATIONAL CONFERENCE OF PHYSICIANS was held in London from September 8th to September 12th. The five sessions of the Section of Cardiology were attended by four or five hundred cardiologists among whom were welcomed many distinguished visitors from overseas. Two of the sessions were joint meetings with the Sections of Pædiatrics and of Disorders of the Chest, respectively. The programme arranged was as follows.

Sept. 9th. Joint Meeting of Sections of Cardiology and Pædiatrics. Discussion on THE SURGERY OF CONGENITAL HEART DISEASE.

Morning: Diagnosis and Treatment of the Cyanotic Group.

Dr. Helen Taussig (U.S.A.)
Dr. Alfred Blalock (U.S.A.)
Dr. James W. Brown (Great Britain)
Contributions to the discussion were made by Prof. C. Laubry, Prof. J. McMichael, and Dr. F. N. Wilson.

Afternoon: The Surgery of Patent Ductus Arteriosus and of Coarctation.

Dr. C. Crafoord (Sweden)
Dr. Rae Gilchrist (Great Britain)
Mr. Holmes Sellors (Great Britain)
Mr. O. S. Tubbs (Great Britain)
Dr. G. Biörck, Prof. C. Bramwell, Mr. Price Thomas, Dr. M. Campbell, Dr. S. Suzman, and Dr. I. Lindgren also spoke.

Sept. 10th. Morning: Joint Meeting with Section of Chest Diseases. Discussion on PULMONARY HEART FAILURE: ACUTE AND CHRONIC.

Prof. J. McMichael (Great Britain)
Dr. J. Lenègre (France)
Dr. W. D. W. Brooks (Great Britain)
Dr. Paul Wood (Great Britain)
The following members also spoke: Dr. C. Lian, Dr. F. N. Wilson, Dr. Fletcher, Dr. Hammarström, and Dr. G. Biörck.

Afternoon: Discussion on THE CLINICAL VALUE OF CHEST LEADS.

Dr. F. N. Wilson (U.S.A.)
Dr. C. W. Curtiss Bain (Great Britain)
Dr. Terence East (Great Britain)
Contributions were also made by Dr. I. G. W. Hill, Prof. C. Lian, and Dr. Van Dooren.

Sept. 11th. Discussion on THE USE OF THE PHONOCARDIOGRAM IN CLINICAL CARDIOLOGY.

Dr. William Evans (Great Britain)
Dr. C. Lian (France)
M. Minot (France)
Prof. C. Bramwell and Dr. Evan Bedford also spoke.

Sept. 12th. Communications.

1. The Septal Anastamoses of the Coronary Arteries.
   Prof. C. Laubry (France)
   Dr. Evan Bedford also spoke.

2. Repetitive Paroxsysmal Tachycardia.
   Dr. John Parkinson and Dr. C. Papp (Great Britain)
   Contributions to the discussion were made by Dr. F. N. Wilson, Dr. C. Lian, Dr. Lenègre, and Prof. C. Laubry.

3. Sympathectomy for Hypertension: preliminary experiences.
   Dr. Geoffrey Bourne (Great Britain)
   Mr. E. G. Tuckwell (Great Britain)
   Dr. F. N. Wilson and Dr. Hammarström contributed to the discussion.

4. Tomography in the study of Cardiovascular Disease.
   Dr. E. Tiscenco (Great Britain)
   Dr. J. H. Wright (Great Britain)

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